JACC Vol. 32, No. 3
September 1998:840–64

ACC EXPERT CONSENSUS DOCUMENT

Present Use of Bedside Right Heart Catheterization in Patients With Cardiac Disease

WRITING GROUP MEMBERS

HILTRUD S. MUELLER, MD, FACC, CHAIR
KANU CHATTERJEE, MD, FRCP, FACC, KATHRYN B. DAVIS, PHD, FACC, MICHAEL A. FIFER, MD, FACC, CORY FRANKLIN, MD, FACC, MARK A. GREENBERG, MD, FACC, ARTHUR J. LABOVITZ, MD, FACC, PREDIMAN K. SHAH, MD, FACC, KENNETH J. TUMAN, MD, FCCM, MAX HARRY WEIL, MD, PHD, FACC, WILLIAM S. WEINTRAUB, MD, FACC

TECHNOLOGY AND PRACTICE EXECUTIVE COMMITTEE

JAMES S. FORRESTER, MD, FACC, CHAIR
PAMELA S. DOUGLAS, MD, FACC, DAVID P. FAXON, MD, FACC, JOHN D. FISHER, MD, FACC, RAYMOND J. GIBBONS, MD, FACC, JONATHAN L. HALPERIN, MD, FACC, JUDITH S. HOCHMAN, MD, FACC, ADOLPH M. HUTTER, JR., MD, MACC, SANJIV KAUL, MD, FACC, WILLIAM S. WEINTRAUB, MD, FACC, WILLIAM L. WINTERS, JR., MD, MACC, MICHAEL J. WOLK, MD, FACC

Contents

Statement of Technology Practice Executive Committee

On September 18, 1996, a report describing a potential increase in morbidity and mortality associated with the use of the pulmonary artery balloon catheter in critically ill patients was published in the *Journal of the American Medical Association* (1). The publication of this report was accompanied by a call for a moratorium on the use of the pulmonary artery balloon catheter until its effectiveness could be documented in appropriately conducted trials, particularly, randomized studies in critically ill patients (2). The American College of Cardiology (ACC) subsequently received a number of requests for statements and clarifications of its position on the use of the pulmonary artery

This Expert Consensus Document was approved by the American College of Cardiology Board of Trustees in March 1998.

Address for reprints: Educational Services, American College of Cardiology, 9111 Old Georgetown Road, Bethesda, Maryland 20814-1699.

©1998 by the American College of Cardiology
Published by Elsevier Science Inc.

0735-1097/98/$19.00
PII S0735-1097(98)00327-1

balloon catheter. In response to these requests, the Technology and Clinical Practice Executive Committee (TPEC) of the ACC recommended to its Board of Trustees in November 1996 that an Expert Consensus Document be developed in response to this question. The TPEC appointed a panel of experts to provide peer comments on the study by Connors et al (1), and to reassess the role of bedside right heart catheterization in patients with cardiac disease. This Expert Consensus Document underwent anonymous external review and subsequent review by both the TPEC and the Board of Trustees of the ACC. The document was approved for publication in March 1998.

JACC Vol. 32, No. 3
September 1998:840–64

Present Use of Bedside Right Heart Catheterization in Patients With Cardiac Disease

Executive Summary

Bedside right heart catheterization (RHC) has been an integral part of cardiovascular practice since the early 1970s. Advances in diagnostic and treatment strategies, including improved noninvasive imaging by Doppler echocardiography and more prevalent use of coronary revascularization by pharmacologic and catheter-based techniques, have changed the role of RHC. A recent case-control study by Connors et al. (1) questioned the safety and efficacy of RHC. The Expert Consensus Committee, convened to develop this document, was charged with two related tasks: 1) to provide peer comments on the study by Connors et al. (1); and 2) to reassess the role of RHC in patients with cardiac disease, providing recommendations for current use and future research. There is a paucity of objective information, particularly of outcome data, derived from clinical trials. There is considerable variation among physicians and nurses in the quality of acquisition and interpretation of the data obtained. These differences create certain difficulties regarding recommendations for the use of RHC, as well as for the planning of multicenter clinical trials. They also emphasize the need for education of physicians and other health professionals in the use of RHC.

Evaluation of case-control study by Connors et al. (1). The Expert Consensus Committee addresses the following issues:

1) RHC is a diagnostic and monitoring device and as such potentially affects outcome only to the extent that it triggers an intervention. Computed tomographic scanning in patients with closed head trauma may well select a population at greater risk but does not confer a higher mortality because of the diagnostic procedure itself. 2) Unexplained clinical factors that prompt clinicians to perform RHC may contribute to the risk of mortality and increased resource consumption. The decision to perform RHC is itself a marker that identifies severely ill patients. Although Connors et al. (1) attempted to eliminate what they term treatment selection bias, the Committee cannot conceive of any method, with the possible exception of randomization, by which such selection bias might be eliminated. Moreover, the computation of the propensity score did not include a measure of responses to therapy before the performance of RHC. 3) The effect of unmeasured confounders was miscalculated, thus overstating the statistical impact required for a variable to overcome the increased mortality. 4) Patients analyzed by Connors et al. (1) may not be characteristic of patients undergoing RHC. Eighty-four percent had either acute respiratory failure or multiorgan failure, neither of which is a specific diagnosis or entity that has a specific treatment and both of which are associated with high mortality.

None of these criticisms vitiates the possibility that there are adverse effects of RHC; however, detailed examination of the data does not provide justification for a moratorium on RHC. The Expert Consensus Committee is concerned that multicenter randomized studies are unlikely to resolve the issues raised by Connors et al. (1) because of the difficulty of standardizing protocols sufficiently to control effects of therapeutic interventions and to accommodate the very large number of patients that would have to be enrolled.

Indications and recommendations for use of bedside RHC. For specific recommendations, see Table 1.

Heart failure. RHC is useful for distinguishing between a cardiogenic and a noncardiogenic (hypovolemic, distributive) mechanism in patients with shock and between a hemodynamic and a permeability mechanism in patients with pulmonary edema. In these settings, the accuracy of clinical assessment of the presence of left heart failure is limited. Accuracy in distinguishing between cardiogenic and noncardiogenic shock and between hemodynamic and permeability pulmonary edema is critical because the management strategies for these conditions are quite distinct. The information provided by RHC often results in a change in therapy. In some patients with shock, a therapeutic trial of volume infusion may be indicated; in others, such empiric therapy may be associated with substantial risk. Similarly, in some patients with dyspnea or pulmonary edema, or both, a trial of diuretic or vasodilator therapy, or both, may be attempted without previous RHC; in others, a strategy that would reduce preload may have deleterious consequences. RHC is warranted when these initial strategies are contraindicated or fail or when there are coexisting manifestations of "forward" and "backward" heart failure. Similarly, RHC is indicated to determine the hemodynamic contribution to respiratory failure in complex patients with concurrent pulmonary and cardiac disease. RHC may be useful for efficiently titrating the dosages of diuretic, vasodilator, inotropic and pressor medications in patients with shock or severe decompensated heart failure, or both. In contrast, RHC is not warranted for the routine management of pulmonary edema, even if endotracheal intubation and mechanical ventilation have been necessary.

RHC is helpful in the management of patients with cardiac disease and failure who are undergoing noncardiac surgery,

Table 1. Recommendations for Use of Bedside Right Heart Catheterization*

Conditions In Which There Is General Agreement That RHC is Warranted	Conditions in Which Reasonable Differences of Opinion Exist Regarding RHC	Conditions in Which RHC Is Not Warranted
Heart Failure		
1. Differentiation between hemodynamic and permeability pulmonary edema or dyspnea (or determination of contribution of left heart failure to respiratory insufficiency in patients with concurrent cardiac and pulmonary disease) when a trial of diuretic and/or vasodilator therapy has failed or is associated with high risk 2. Differentiation between cardiogenic and noncardiogenic shock when a trial of intravascular volume expansion has failed or is associated with high risk; guidance of pharmacologic and/or mechanical support 3. Guidance of therapy in patients with concomitant manifestations of "forward" (hypotension, oliguria, and/or azotemia) and "backward" (dyspnea and/or hypoxemia) heart failure 4. Determination of whether pericardial tamponade is present when clinical assessment is inconclusive and echocardiography is unavailable, technically inadequate or nondiagnostic 5. Guidance of perioperative management in selected patients with decompensated heart failure undergoing intermediate or high risk (3) noncardiac surgery 6. Detection of presence of pulmonary vasoconstriction and determination of its reversibility in patients being considered for heart transplantation	1. Differentiation between hemodynamic and permeability pulmonary edema or dyspnea (or determination of the contribution of left heart failure to respiratory insufficiency in patients with concurrent cardiac and pulmonary disease) when a trial of diuretic and/or vasodilator therapy is associated with low or intermediate risk 2. Differentiation between cardiogenic and noncardiogenic shock when a trial of intravascular volume expansion is associated with intermediate risk 3. Facilitation of titration of diuretic, vasodilator and inotropic therapy in patients with severe heart failure 4. Guidance of perioperative management in patients with compensated heart failure undergoing intermediate or high risk (3) noncardiac surgery	1. Routine management of pulmonary edema, even if endotracheal intubation and mechanical ventilation have been necessary 2. Differentiation between cardiogenic and noncardiogenic shock before a trial of intravascular volume expansion, when such a trial is associated with low risk 3. Institution or titration of diuretic and/or vasodilator therapy in patients with mild or moderate heart failure 4. Marked hemodynamic instability in patients in whom pericardial tamponade is certain or probable by clinical and/or echocardiographic criteria and RHC would delay treatment 5. Guidance of perioperative management in patients with compensated heart failure undergoing low risk (3) noncardiac surgery
Acute Myocardial Infarction		
1. Differentiation between cardiogenic and hypovolemic shock when initial therapy with intravascular volume expansion and low doses of inotropic drugs has failed 2. Guidance of management of cardiogenic shock with pharmacologic and/or mechanical support in patients with and without coronary reperfusion therapy 3. Short-term guidance of pharmacologic and/or mechanical management of acute mitral regurgitation (with or without disruption of the mitral valve) before surgical correction 4. Establishment of severity of left to right shunting and short-term guidance of pharmacologic and/or mechanical management of ventricular septal rupture before surgical correction 5. Guidance of management of right ventricular infarction with hypotension and/or signs of low cardiac output not responding to intravascular volume expansion, low doses of inotropic drugs and/or restoration of heart rate and atrioventricular synchrony 6. Guidance of management of acute pulmonary edema not responding to treatment with diuretic drugs, nitroglycerin, other vasodilator agents and low doses of inotropic drugs	1. Guidance of ongoing management of hypotension, *after* response to initial therapy with intravascular volume expansion and/or low doses of inotropic drugs 2. *Short-term* guidance of pharmacologic and/or mechanical management of acute mitral regurgitation if operation is delayed or not contemplated 3. Establishment of severity of left to right shunting and *short-term* guidance of pharmacologic and/or mechanical management of ventricular septal rupture if operation is delayed or not contemplated 4. Guidance of management of right ventricular infarction, *after* correction of hypotension and/or signs of low cardiac output by intravascular volume expansion, low doses of inotropic drugs and/or restoration of heart rate and atrioventricular synchrony 5. Guidance of management of acute pulmonary edema with vasodilators and/or inotropic drugs, *after* initial treatment with diuretic drugs and nitroglycerin has failed 6. Confirmation of diagnosis of pericardial tamponade subsequent to subacute myocardial rupture when clinical and echocardiographic assessments are inconclusive	1. Guidance of management of postinfarction angina 2. Guidance of ongoing management of pulmonary edema responding promptly to treatment with diuretic drugs and nitroglycerin 3. Pericardial tamponade with marked hemodynamic instability, when the diagnosis is certain or likely by clinical and/or echocardiographic criteria and RHC would delay treatment

(Continued)

Table 1. (continued)

Conditions In Which There Is General Agreement That RHC is Warranted	Conditions in Which Reasonable Differences of Opinion Exist Regarding RHC	Conditions in Which RHC Is Not Warranted
Perioperative Use in Cardiac Surgery		
1. Differentiation between causes of low cardiac output (hypovolemia vs. ventricular dysfunction), when clinical and/or echocardiographic assessment is inconclusive 2. Differentiation between right and left ventricular dysfunction and pericardial tamponade, when clinical and/or echocardiographic assessment is inconclusive 3. Guidance of management of severe low cardiac output syndrome 4. Diagnosis and guidance of management of pulmonary hypertension in patients with systemic hypotension and evidence of inadequate organ perfusion	1. Guidance of inotropic and/or vasopressor therapy, *after* patients with significant cardiac dysfunction have achieved hemodynamic stability 2. Guidance of management of hypotension and evidence of inadequate organ perfusion when a therapeutic trial of intravascular volume expansion and/or vasoactive agents is associated with moderate risk	1. Routine management of uncomplicated cardiac surgical patients with good ventricular function and hemodynamic stability 2. Initial management of postoperative hypotension when a therapeutic trial of volume expansion and/or vasoactive agents is associated with low risk
Primary Pulmonary Hypertension		
1. Exclusion of postcapillary (elevated PAOP) causes of pulmonary hypertension 2. Establishment of diagnosis and assessment of severity of precapillary (normal PAOP) pulmonary hypertension 3. Selection and establishment of safety and efficacy of long-term vasodilator therapy based on acute hemodynamic response 4. Assessment of hemodynamic variables before lung transplantation	1. Evaluation of long-term efficacy of vasodilator therapy, particularly prostacyclin 2. Exclusion of significant left to right or right to left intracardiac shunt	None

*Relative and absolute contraindications, see Main document below.

particularly patients with decompensated heart failure undergoing intermediate or high risk operation. RHC aids in risk stratification of patients who are considered for heart transplantation. Patients with substantial fixed elevation of pulmonary vascular resistance have a poor prognosis; patients with reversible pulmonary vasoconstriction have a more favorable outcome. Preoperative RHC identifies patients with high pulmonary vascular resistance and determines its reversibility in response to vasodilator agents.

Pericardial tamponade constitutes a special case of the diagnosis and management of heart failure. Tamponade is suspected on clinical grounds and is best confirmed by echocardiography. RHC is warranted when clinical and echocardiographic findings are equivocal. In hemodynamically unstable patients with suspected pericardial tamponade, timely therapy should not be delayed by the performance of RHC.

Acute myocardial infarction. The use of RHC in acute myocardial infarction with hemodynamic instability depends on the underlying cause and the course of hemodynamic compromise. In patients with either transient hypotension or hypotension that responds promptly to intravascular volume expansion and/or low doses of inotropic drugs, RHC is not required. If patients do not improve rapidly, RHC is warranted to distinguish cardiogenic from other forms of shock and to guide the assessment of the *short-term* response to pharmacologic agents and mechanical support (intraaortic balloon pumping). Similarly, transient hypotension and clinical findings of low cardiac output in the setting or right ventricular infarction can often be managed without RHC, using cautious volume loading, small doses of inotropic drugs and, if required, temporary pacing. RHC is warranted when hemodynamic compromise persists despite initial therapy. Careful monitoring of cardiac output is important because ventricular interaction and pericardial constraint observed in right ventricular infarction alter diastolic properties of the left ventricle such that pulmonary artery occlusive pressure (PAOP) is not a reliable indicator of left ventricular preload.

In the setting of hemodynamic instability caused by acute mechanical complications after acute myocardial infarction, echocardiography with Doppler imaging is the primary procedure for the diagnosis. Acute mitral regurgitation with or without valve disruption, ventricular septal rupture and pericardial effusion or tamponade can be diagnosed rapidly by this modality. The main indications for RHC in these settings relate to short-term hemodynamic monitoring of preoperative and postoperative pharmacologic and mechanical support. RHC is helpful for monitoring the effect of therapeutic interventions on the magnitude of shunting through the ventricular septal defect.

The use of RHC for the guidance of pharmacologic therapy in patients with heart failure or pulmonary edema (including those requiring mechanical ventilation), and for prolonged monitoring of patients with persistent hemodynamic instability who are not considered for coronary revascularization or

surgical repair of structural complications, represent intermediate indications for which considerable disagreement exists.

Perioperative use in cardiac surgery. Patients undergoing cardiac surgery may develop disturbances of perfusion that can be life-threatening and/or place the function of vital organs at risk. Identification of the underlying hemodynamic derangements determines therapeutic strategy. RHC facilitates both the diagnosis and management of low cardiac output states that may not always be adequately assessed by clinical markers of perfusion after cardiac surgery. Identification of abnormalities of cardiac output as well as right and left ventricular afterload or preload is particularly important to guide pharmacologic and other therapy aimed at optimizing perfusion. Finally, cardiac surgical techniques involving cardiopulmonary bypass may result in the accumulation of extravascular lung water, and RHC often serves as a useful guide for evaluating the effects of fluid management on global variables of perfusion after cardiac surgery.

Available data do not support the routine use of RHC in the perioperative period in the hemodynamically stable patient with good ventricular function. RHC is useful in patients undergoing cardiac surgery with previous evidence of ventricular dysfunction of any cause, complex coronary artery disease or valvular disease (especially when it is associated with significant coronary artery disease or pulmonary hypertension) and in patients requiring repeat cardiac surgery.

In the postoperative period, RHC is warranted for differentiation among causes of low cardiac output when clinical and echocardiographic evaluations are inconclusive or therapeutic trials of intravascular volume expansion and vasoactive agents have failed or are associated with high risk. However, reasonable differences of opinion exist as to whether RHC should be used for guidance of inotropic and/or vasopressor therapy after hemodynamic stability has been achieved in patients with significant cardiac dysfunction.

Primary pulmonary hypertension. RHC is warranted for establishing the diagnosis of precapillary (normal PAOP) pulmonary hypertension and to accurately assess its severity. Although echocardiography/Doppler imaging can be used to estimate pulmonary artery systolic pressure, measurement of PAOP, which is essential to distinguish between postcapillary and precapillary pulmonary hypertension and to determine pulmonary vascular resistance, cannot presently be accomplished without RHC. RHC is warranted before institution of long-term pharmacologic therapy of primary pulmonary hypertension, in particular when calcium channel blocking agents are used, to evaluate the hemodynamic response to vasodilators. The pattern of the acute hemodynamic response has been shown to correlate with long-term prognosis. Adverse hemodynamic responses, such as a reduction in cardiac output or systemic arterial pressure, or an increase in right atrial pressure, regarded as contraindications to long-term vasodilator therapy, can only be determined by hemodynamic monitoring. There is considerable difference of opinion as to whether RHC should be used for evaluation of long-term efficacy of vasodilator therapy.

Complications. Complications related to RHC are associated with the establishment of central venous access, the catheterization procedure itself and catheter residence.

Adverse events related to central venous cannulation include arterial puncture, bleeding at the site of insertion, nerve injury, pneumothorax and air embolism. The primary complications related to the RHC procedure itself are arrhythmias, which are usually clinically insignificant. Sustained ventricular arrhythmias are uncommon and occur primarily in patients with myocardial ischemia or infarction or preexisting ventricular arrhythmias. The most serious complications of RHC are related to catheter residence. They include pulmonary artery rupture, thrombophlebitis, venous or intracardiac thrombus formation, pulmonary infarction and endocarditis. The risk of thrombotic and infectious complications increases significantly when the catheter remains in place >3 to 4 days.

Alternative or complementary procedures. Echocardiography with Doppler imaging is the most important alternative or complementary procedure to RHC in the critically ill cardiac patient. It elucidates the likely mechanism of hypotension and shock, aiding the distinction between cardiogenic and noncardiogenic shock. Echocardiography/Doppler imaging has a primary role in the diagnosis of mechanical complications after acute myocardial infarction, including acute mitral regurgitation with and without valve disruption, ventricular septal defect and ventricular free wall rupture. Transesophageal echocardiography (TEE) is superior to transthoracic echocardiography (TTE) in certain patients, facilitating data acquisition and enhancing diagnostic information. Estimation of left ventricular filling pressure and stroke volume for hemodynamic monitoring has not yet been validated for widespread clinical use as an alternative to RHC. These techniques remain the subject of continued clinical investigation. TEE plays an important role in the perioperative period for evaluation of left ventricular function and results of valve repair. It is of particular value in the patient with hypotension and low cardiac output during separation from cardiopulmonary bypass. A variety of promising developments, including miniaturized probes and techniques for real time on-line monitoring of ventricular volumes, ejection fraction and compliance, are under clinical investigation.

Proposals for randomized trials. The Expert Consensus Committee considered carefully the role of future research for the evaluation of the safety and efficacy of RHC in patients with cardiac disease. It identified two areas, namely heart failure and cardiac surgery, in which RHC is frequently used and randomized trials may be considered. The Committee came to the following conclusions:

1. Randomized clinical trials in patients with heart failure are ethical in those subjects (such as patients with severe heart failure who require titration of diuretic, vasodilator and inotropic therapy) for which reasonable differences of opinion exist regarding the indication for RHC. For considerations of sample size and power, end points other than mortality would be necessary and should include morbidity, length of intensive care unit (ICU) and total hospital stay and cost. For the benefit

of RHC to be assessed, it must be deployed in clinical conditions for which beneficial treatment is available.

2. The efficacy and safety of RHC in *low risk* patients undergoing cardiac surgery could be established by a clinical trial, but such a trial would be difficult to perform. RHC would be expected to have minimal effect on mortality and morbidity, and the required sample size would be very large. In the absence of sufficient power for examining a clinical end point, a cost-effectiveness study would not be feasible. In the case of an equivalent outcome whether or not RHC was used, a pure cost study (cost minimization study) would be possible. A clinical trial in *high risk* patients undergoing cardiac surgery is more likely to be revealing because the incidence of end points will be higher, and the potential of RHC to affect outcome would appear to be greater than in a trial in low risk patients. However, the Expert Consensus Committee is concerned that such a trial would be difficult to perform because of unwillingness of physicians to randomize patients.

Conclusions. The Expert Consensus Committee was convened to formulate recommendations for the use of bedside RHC in patients with known or suspected cardiac disease. Because few data are available from well controlled clinical trials, the recommendations are based primarily on inferences from published reports and expert opinion. The role of RHC is currently in transition because of the availability of noninvasive diagnostic techniques, in particular, echocardiography with Doppler, which in some cases complement and in others are alternatives to RHC. RHC has a primary role in the management of heart failure in certain patients in whom the diagnosis of heart failure is uncertain, heart failure is of sufficient severity to require intensive pharmacologic management or heart transplantation is contemplated. In patients with acute myocardial infarction, echocardiography has a primary role in the diagnosis of mechanical complications, and RHC is helpful in certain instances for assessment of the severity of hemodynamic compromise and the response to therapy. The Committee does not recommend the routine use of RHC in patients undergoing uncomplicated cardiac surgery who have good ventricular function and hemodynamic stability.

The Committee is not optimistic about the feasibility of randomized clinical trials to establish the safety and efficacy of RHC, with the possible exception of a trial in patients with refractory decompensated heart failure. The Committee questions whether major resources should be devoted to reevaluation of the role of RHC or whether such resources would be better directed toward future development and evaluation of semi-invasive and noninvasive diagnostic techniques.

Present Use of Bedside Right Heart Catheterization in Patients With Cardiac Disease

Preamble

Method of topic selection. This Expert Consensus Document on right heart catheterization (RHC) at the bedside was developed at the request of the Technology and Practice Executive Committee of the American College of Cardiology (ACC) in response to a major increase in interest in this area. The Expert Consensus Document is intended to inform practitioners, payers and other interested parties of the opinion of the ACC concerning evolving areas of clinical practice and/or technologies that are widely available or are new to the practice community. Topics chosen for coverage by Expert Consensus Documents are so designated because the evidence base and experience with the technology or clinical practice are not sufficiently well developed to be evaluated by the formal ACC/American Heart Association (AHA) practice guidelines process. Thus, the reader should view the Expert Consensus Document as the best attempt of the ACC to inform and guide clinical practice in areas where rigorous evidence is not yet available. Some topics covered by Expert Consensus Documents will be addressed subsequently by the ACC/AHA practice Guidelines process.

Introduction

The introduction in the early 1970s of bedside RHC (4) led to profound changes in the practice of cardiology and fostered major advances in the diagnosis and treatment of critically ill patients with cardiac disease. However, new cardiac diagnostic and treatment strategies that have evolved in the past two decades have placed RHC in a different perspective. Improved imaging modalities, in particular, TTE and TEE, provide important information regarding cardiac function and structure frequently in lieu of RHC. The increased use of cardiac catheterization, including RHC, and mechanical coronary revascularization in patients with acute ischemic syndromes has decreased the need for bedside RHC. New pharmacologic agents, such as thrombolytic agents and antiplatelet drugs, present relative contraindications to RHC and thus favor noninvasive assessment over RHC.

A recent case-control study (1) reported excess mortality and length of stay in a diverse group of cardiac and noncardiac patients who had undergone RHC, and an accompanying editorial called for a moratorium on RHC (2). In response to these publications, the Technology and Practice Executive Committee (TPEC) appointed this Expert Consensus Committee and charged it with two related tasks: 1) to provide peer comments on the study by Connors et al. (1); and 2) to reassess the role of RHC in patients with cardiac disease, providing recommendations for current use and future research. The Consensus Committee included a variety of experts, encompassing invasive and noninvasive cardiology, critical care medicine, cardiac anesthesia, epidemiology and biostatistics. The recommendations of the Committee are based on published data and, in the absence of conclusive data, on a consensus among its members. In accordance with ACC policy, the recommendations are prefaced by background information regarding the various clinical situations considered by the Committee.

Several societies have established guidelines for the use of RHC, including recommendations for quality control (5–10). Each of the guidelines recognized the paucity of objective information, particularly outcome data, derived from randomized trials. Although prospective, randomized trials do form a basis for clinical practice, any valid study of RHC requires a definable measure of competency in both the acquisition and interpretation of data from RHC. There is considerable variation among physicians (11,12) and nurses (13) in the quality of acquisition and interpretation of the data obtained by RHC and subsequent integration into therapeutic strategies. There is also considerable variation in the experience of physicians caring for critically ill patients with cardiac disease and in the perceived need for RHC. These differences create certain difficulties regarding recommendations for the use of RHC as well as for the planning of multicenter trials. Programs for education of physicians and other health professionals in the use of RHC are of considerable importance.

The design of future research on the role of bedside RHC should consider alternative monitoring options that are less invasive. Echocardiographic acquisition of anatomic and physiologic data (14), Doppler estimation of cardiac pressures (15,16) and continuous TEE in the critical care setting (17,18) are presently under intense clinical investigation and may evolve to promising tools for the provision of comprehensive information. Careful consideration is required to determine whether resources should be directed mainly toward the reevaluation of bedside RHC or should be directed preferentially toward the development and investigation of newer noninvasive or less invasive monitoring modalities.

Evaluation of a Case-Control Study of RHC by Connors Et Al.

The recent study by Connors et al. (1) concluded that after adjustment for treatment selection bias, RHC was associated with increased mortality and increased utilization of resources. An accompanying editorial proposed a moratorium on RHC (2). The Expert Consensus Committee addressed the following issues:

The right heart catheter is a monitoring device and as such is a diagnostic rather than a treatment modality. In life-threatening settings, the overriding benefit of a diagnostic or monitoring device is the intervention that it triggers. In this respect, the adverse impact of a diagnostic intervention, including the potential morbidity and, in rare instances, mortality stemming from the use of the device itself, cannot be evaluated except to the extent that therapeutic interventions are controlled. For example, request of computed tomographic scans for patients with closed head trauma may well select a population at greater risk, but it does not imply a higher mortality for them because they undergo risks of transport and positioning for imaging. The objective documentation in the report of Connors et al. (1) therefore does not allow for clear-cut separation of any adverse effect of the diagnostic intervention, represented by RHC, and the severity of the disorder together with the effects of a diversity of therapeutic interventions. The observations reported by Connors et al. (1) are alternatively explained by selection of more serious disorders and unsuccessful treatment strategies (19).

Unexplained clinical factors that prompt clinicians to perform RHC may contribute significantly to the risk of mortality and increased resource consumption. The decision to perform RHC is itself a marker that identifies severely ill patients. We recognize the attempts of Connors et al. (1) to eliminate what they term *treatment selection bias*. Yet, if this diagnostic intervention selects patients for whom there is greater likelihood of morbidity and mortality, we cannot conceive of any method, with the possible exception of randomization, by which such selection might be eliminated. Because the study by Connors et al. (1) was not randomized, comparisons between patients who did and those who did not receive RHC depended on a propensity score intended to predict whether RHC would be utilized. Among the clinical variables included in the computation of the propensity, responses to therapy were excluded. To emphasize this point, the clinical features of two patients presenting with pulmonary edema might be considered. The patient responding to initial therapy with diuretic drugs would probably not become a candidate for RHC, whereas the patient in whom more extensive pharmacologic therapy failed would be more likely to undergo RHC, thus inevitably facing a higher mortality risk (20,21). Yet the propensity score as presented would fail to distinguish between these patients.

The effect of unmeasured confounders was miscalculated. Connors et al. (1) estimated that to account for the 20% increased risk of death in the RHC group, a missing covariate would have to increase the risk of death and the probability of RHC threefold. To change the 20% increased risk to a 20% decreased risk, a missing covariate would have to increase the risk of death and the probability of RHC sixfold. In calculating these estimates, Connors et al. (1) have used their estimated odds ratios as if they were probability ratios. An increase in the probability of RHC increases the odds ratio more than it increases the probability ratio. Thus, the effect of unmeasured confounders needed to reduce the observed association was overestimated. The effect that would change the observed 20% increased risk in the RHC groups to no increased risk is actually only a twofold increase in propensity and mortality. A threefold increase in the probability of RHC and the risk of death would change the relative risk to a 20% decrease in risk (22). Thus, an unmeasured confounder with only a moderately independent effect could account completely for the observed association.

The development of the propensity score and the sensitivity analysis apply primarily to patients with acute respiratory and multiple organ system failure. Patients with acute respiratory and multiple organ system failure account for 80% of the patients analyzed by Connors et al. (1). No patients with cardiac diagnoses, possibly the largest group undergoing RHC in clinical practice, were included in these groups. In the 11% of patients who had congestive heart failure (CHF), the relative odds of death was 1.02, indicating neither harm nor benefit. The addition of an unmeasured covariate with only a modest effect on propensity and mortality could change the estimated odds in this group to significant benefit for RHC. Possible benefit for other patient subgroups included in the largest groups may also have been overlooked.

None of these specific criticisms vitiates the possibility that there are adverse effects of RHC. However, a detailed examination of the data does not provide justification for a moratorium on RHC. More objective information is needed before any such drastic action can be recommended (9,11). The Expert Consensus Committee is concerned that multicenter, randomized studies are unlikely to resolve these issues because of the difficulty of standardizing protocols sufficiently to control effects of therapeutic interventions and to accommodate the very large number of patients that would have to be enrolled. Because critically ill patients are a heterogeneous group, subgroups would have to be defined. The challenge of achieving adequate statistical power in such subgroups is likely to be very great. Furthermore and perhaps more fundamentally, the use of RHC is not in equipoise (23,24).

Although the Committee cautions against complacency, it believes that it is crucial, in the absence of scientifically secured data, to maintain objectivity and secure appropriate consensus with respect to the questions posed by Connors et al. (1).

Acquisition and Interpretation of Data

Safe and effective use of RHC is predicated on careful catheter placement, attention to measurement techniques and thoughtful interpretation of the data. Skillful placement of the

catheter and interpretation of data will optimize the benefit/risk ratio of this modality.

RHC must be performed with strict sterile technique. If the catheter is placed through a protective sleeve with meticulous attention to maintaining its sterility, the catheter may be advanced, if necessary, within 24 h of placement. After 24 h, the catheter should preferably not be advanced. To minimize the risk of infection, the catheter should be left in place for only as long as it provides information essential for patient management. In general, this period should not exceed 3 days; when catheters are left in place for >3 days, justification should be provided. The strategy of routine replacement of catheters over guide wires or through repeat venipuncture has not been demonstrated to reduce the risk of infection (25).

When the catheter tip is in the pulmonary artery, the balloon should be inflated slowly, using tactile assessment of balloon pressure and intravascular pressure waveform monitoring; this will minimize the risk of pulmonary artery rupture and associated hemorrhage. If concordance between pulmonary artery occlusive pressure (PAOP) and pulmonary artery diastolic pressure is established, frequent measurements of the former may not be necessary.

In most cases, RHC through the internal jugular, subclavian or antecubital approach may be performed at the bedside, without fluoroscopy. Fluoroscopic guidance should be considered in the presence of a temporary or recently placed permanent pacemaker or implantable cardioverter-defibrillator; right atrial and/or ventricular dilation, severe tricuspid regurgitation; or left bundle branch block. In the latter instance, fluoroscopy offers the advantages of minimizing catheter manipulation in the right heart, lessening the likelihood of concurrent right bundle branch block leading to complete block, and of facilitating the rapid positioning of a temporary pacemaker, should complete heart block nevertheless occur. Alternatively, the availability of standby transcutaneous pacing allows for RHC in the presence of left bundle branch block without the use of fluoroscopy.

RHC allows for measurement of 1) central venous or right atrial pressure; 2) pulmonary artery systolic, diastolic and mean pressures; 3) PAOP or "wedge" pressure; 4) thermodilution cardiac output; and 5) oxygen saturation.

Current practice is to measure right heart pressures relative to zero pressure defined at the midaxillary line. It has been suggested that pressure is more correctly referenced from the upper border of the left ventricle (26) and that zero reference pressure is best estimated in a plane 5 cm below the sternal angle (27). Meticulous flushing of catheters, transducer devices and tubing to remove blood and air reduces the risk or errors in measurement caused by damping of pressure tracings.

According to principles of cardiopulmonary physiology, current ICU practice is to record PAOP and other pressures at end-exhalation. This practice differs from that of cardiac catheterization laboratories (and in a large body of published reports on which much of our understanding of the clinical significance of pressure measurements is based), which is to record mean pressures averaged throughout the respiratory cycle. These disparate practices create a discrepancy between pressures measured in the ICU and those measured in the cardiac catheterization laboratory. In a patient breathing without the aid of positive pressure, end-exhalatory pressure is higher than mean pressure; in a patient receiving positive pressure ventilation, end-exhalatory pressure is lower than mean pressure. Until the practices of ICUs and cardiac catheterization laboratories converge, the clinician must be aware of the differences in measurement technique and must interpret the data accordingly.

For the PAOP to reflect left atrial pressure accurately, there must be a patent fluid column between the catheter tip and the left atrium. If alveolar pressure exceeds hydrostatic pressure in the pulmonary capillaries, they collapse; in this instance, the measured pressure may be grossly misleading. Thus, the pressure recorded when a pulmonary artery branch is occluded by a balloon (PAOP) reflects pulmonary venous (and thus left atrial) pressure only when pulmonary venous (P_v) and pulmonary artery (P_a) pressures exceed pulmonary alveolar (P_A) pressure (i.e., $P_a > P_v > P_A$, defined as West zone 3 [28]). In other regions of the lung (West zones 1 and 2), balloon inflation results in measurement of alveolar pressure (29). Fortunately, with the patient supine, most of the lung consists of zone 3 units (30).

In the presence of positive end-expiratory pressure (PEEP), alveolar pressure is higher, so that less of the lung consists of zone 3 units, and the likelihood of catheter placement in zones 1 or 2 increases. Inaccurate estimation of left atrial pressure is likely when the catheter is above the level of the left atrium (31) or when the catheter is at the level of the left atrium but the left atrial pressure is low. In one study (32), 43% of catheters placed through the internal jugular approach lodged at or above the level of the left atrium, yielding inaccurate assessment of left atrial pressure in the presence of PEEP. Even in the absence of an increase in alveolar pressure to a level that exceeds pulmonary venous pressure, the use of PEEP may affect the measurement of intravascular pressures because the positive airway pressure may be transmitted to the central vessels. The problem is particularly significant when >10 cm H_2O of PEEP is used (33,34). Although a formula for correction has been offered (35), the extent to which an increase in airway pressure is transmitted to the pulmonary vasculature is variable and not accurately predictable.

Although PAOP is often used as an estimate of both pulmonary capillary pressure, the driving force for shifting fluid from pulmonary capillaries into the interstitium and alveoli, and left ventricular diastolic pressure, it is an imperfect measure of both. Although the pulmonary capillary pressure exceeds PAOP by only a few mm Hg in the normal lung, it may exceed PAOP by 10 to 15 mm Hg in sepsis and other inflammatory disorders, resulting in pulmonary edema despite an "acceptable" PAOP (36). PAOP reflects left atrial pressure, which is in turn indicative of left ventricular diastolic pressure only in the absence of mitral stenosis or more than mild mitral regurgitation. Furthermore, the relation between left ventricular pressure and volume in diastole depends critically on ventricular compliance, which is often abnormal in critically ill patients.

The concept of an "optimal" PAOP for maximizing left ventricular output was introduced along with the pulmonary artery catheter. Traditional values for "optimal" PAOP, typically 14 to 18 mm Hg, were based on early data from patients with acute myocardial infarction (37). Since then there have been changes both in the therapy of myocardial infarction and in the techniques for measuring PAOP. Despite these changes, the concept of optimal PAOP has remained and has been extrapolated to other patient groups. However, it has been shown (38) that effective vasodilator treatment of patients with heart failure results in higher output and lower PAOP, with no "lower limit" or optimal PAOP. Optimal PAOP varies greatly among patients and requires an empiric approach that assesses clinical status and cardiac output at various filling pressures in an individual patient. In assessing the effects of therapeutic interventions, directional changes in PAOP may be more useful than absolute values of PAOP.

Pressures and thermodilution cardiac output measurements should be interpreted, when appropriate, together with measurements of pulmonary artery oxygen saturation and arteriovenous oxygen difference. Thermodilution cardiac output may be inaccurate in the presence of arrhythmias, tricuspid regurgitation (39) and intracardiac shunting. Data obtained from RHC should be interpreted in the context of clinical assessment, taking into account weight, input/output tallies, mental status, skin temperature, lung examination, urine output, blood urea nitrogen, creatinine, arterial Po_2 and pH and chest radiographic and, when appropriate, echocardiographic findings.

Technology of right heart catheters. The routinely used balloon flotation catheters have four lumens, including two for transmission of pressure signals from the pulmonary artery and the right atrium, one for balloon inflation and one for a thermostat located near the catheter tip. Multipurpose electrode catheters incorporate atrial and ventricular electrodes for recording of intraatrial and intraventricular electrocardiograms, facilitating the diagnosis of complex arrhythmias (40) and, in rare instances, temporary pacing. Newer catheters have a fifth lumen containing fiberoptic bundles for measurement of mixed venous oxygen saturation. Balloon flotation catheters with the additional capability of measuring right ventricular stroke volume, end-systolic and end-diastolic volumes and ejection fraction have been introduced (41). Continuous determination of cardiac output is feasible by the thermodilution principle with the use of a specially designed cardiac output computer without requiring delivery of indicator solution (42). Alternatively, Doppler techniques may be used for determination of continuous cardiac output (43). Heparin-bond catheters designed to decrease catheter thrombosis and catheters impregnated with antiseptic agents are also available.

Method of Data Collection

A MEDLINE search of published reports in English from 1966 to 1997 was performed. Swan-Ganz catheterization or RHC in patients was cross-checked with the following terms: unstable angina, myocardial infarction, pulmonary edema, cardiogenic shock, flail mitral leaflet, mitral valve papillary muscle rupture, ventricular septal rupture, pericardium, pericardial effusion, pericardial tamponade, heart failure, decompensated heart failure, cardiac surgery, coronary artery bypass graft surgery (CABG), heart transplantation, pulmonary hypertension, inotropic agents, vasodilator agents, vasoconstrictor agents and intraaortic balloon pumping (counterpulsation). The document took in account this review of the published reports. In addition, members of the Expert Consensus Committee utilized published reports at their own discretion, according to their particular fields of interest and expertise. Guidelines related to cardiology, cardiac surgery, cardiac anesthesiology and critical care medicine published during the past decade were reviewed.

Grading of recommendations. The recommendations were graded according to the guidelines for Expert Consensus Documents provided by the TPEC of the ACC, as follows:

1. Conditions in which there is general agreement that RHC is warranted.
2. Conditions in which reasonable differences of opinion exist regarding RHC.
3. Conditions in which RHC is not warranted.
4. Conditions in which a relative contraindication to RHC exists.
5. Conditions in which an absolute contraindication to RHC exists.

Indications and Recommendations for Use of Bedside RHC

Heart Failure

Background. *Accurate assessment of hemodynamic status.* In patients with shock, the distinction between a cardiogenic and a noncardiogenic (hypovolemic, distributive) mechanism is essential for directing management. In patients with pulmonary edema, the distinction between a hemodynamic and a permeability mechanism determines therapeutic strategy. In these settings, a number of studies have suggested that the accuracy of clinical assessment of the presence of left heart failure is limited. Clinical assessment is handicapped by the fact that the radiographic appearance of pulmonary edema is neither sensitive nor specific for the diagnosis of pulmonary venous hypertension. In particular, patients with chronic elevation of PAOP may have compensatory mechanisms that obfuscate physical and radiographic signs of left heart failure. Thus, in patients with decompensation of chronic heart failure (as distinct from patients with acute myocardial infarction), clinical assessment may be insensitive for the detection of elevated PAOP (44,45). Conversely, the radiographic finding of pulmonary edema is nonspecific for the diagnosis of left heart failure (46); there are many causes of permeability pulmonary edema. Several studies of patients without acute myocardial infarction carried out in the medical ICU setting have suggested that clinical assessment of PAOP may be inaccurate (47,48).

Accuracy in distinguishing between cardiogenic and noncardiogenic shock and between hemodynamic and permeability pulmonary edema is critical because the management strategies for the two conditions are quite distinct. The information provided by RHC often results in a change in therapy (47,48).

Pericardial tamponade. Patients with suspected pericardial tamponade constitute a special case of the diagnosis of heart failure. Tamponade is suspected on clinical grounds (tachycardia, pulsus paradoxus and jugular venous distention) and is best confirmed by echocardiography (pericardial effusion with Doppler echocardiographic signs of tamponade). In some cases, echocardiography may be unavailable or technically suboptimal. The findings of "equalization" of right atrial, right ventricular diastolic, pulmonary artery diastolic pressures and PAOP and of absence or blunting of the y descent in the right atrial tracing by RHC support the diagnosis of pericardial tamponade. Equalization of pressures may occur in other conditions, such as right ventricular infarction and pericardial constriction, and may be absent in pericardial tamponade, for example, if there is an independent cause of elevation of left atrial pressure. In selected patients, RHC may be useful for documenting the hemodynamic response (fall in filling pressures, increase in cardiac output) that accompanies removal of pericardial fluid and for detecting persistently abnormal hemodynamic variables (elevated right atrial pressure with prominent x and y descents) in patients with effusive–constrictive pericardial disease (49). In hemodynamically unstable patients with suspected pericardial tamponade, timely therapy should not be delayed by the performance of RHC.

Management of severe heart failure. Invasive hemodynamic measurements may be useful for effectively and efficiently titrating the dosages of diuretic, vasodilator and inotropic medications, particularly during acute exacerbations of chronic heart failure or hemodynamic instability (50). Stevenson et al. (51) have introduced the concept of "tailored therapy" for heart failure. In their initial report, 50 patients who were candidates for urgent heart transplantation underwent RHC. Nitroprusside infusion and intermittent intravenous furosemide were administered in an attempt to achieve a PAOP of 15 to 20 mm Hg and a systemic vascular resistance $\leq$1,200 dynes·s/cm^5 while maintaining systolic blood pressure $\geq$80 mm Hg. Oral vasodilator agents were subsequently added as intravenous medications were discontinued. Of the 50 patients, 40 were discharged without transplantation (51). In 152 patients undergoing such tailored therapy, mortality was predicted by failure of PAOP to improve during therapy ($p = 0.005$) (52). The authors acknowledged that it is not known whether the achievement of low filling pressures improved survival or merely identified those patients with potential for survival (52). A study of survivors demonstrated continued hemodynamic efficacy of therapy after a mean of 8 months of follow-up (53).

Management of patients with heart failure undergoing noncardiac surgery. The special case of management of patients with heart failure undergoing noncardiac surgery was addressed in the 1996 report of the ACC/AHA Task Force on Practice Guidelines (Committee on Perioperative Cardiovascular Evaluation for Noncardiac Surgery) (3). The Committee suggested that "patients with signs and symptoms of heart failure preoperatively, who have a very high (35%) postoperative incidence of heart failure, may benefit from invasive hemodynamic monitoring." It recommended that "preoperative preparation in an intensive care unit may benefit certain high risk patients, particularly those with decompensated CHF" and those undergoing high risk (emergency, prolonged or certain vascular) operations. The introduction of TEE has decreased the intraoperative use of RHC in some institutions. However, the hemodynamic information obtained from RHC is complementary to that derived from TEE, and RHC provides serial data to guide management in the postoperative period, when information from TEE is less readily available.

Evaluation of patients for heart transplantation. Evaluation for heart transplantation constitutes another special case of the management of patients with heart failure. The outcome of heart transplantation is poor in patients with substantial fixed preoperative elevation of pulmonary vascular resistance; such patients have a high incidence of postoperative right heart failure (54). Patients with reversible pulmonary vasoconstriction have a more favorable outcome after heart transplantation (55). Preoperative RHC identifies patients with high pulmonary vascular resistance and determines reversibility in response to vasodilator agents, such as oxygen, nitroprusside and nitric oxide (56). This assessment may be carried out in the ICU or cardiac catheterization laboratory.

Expert opinion of the Committee. Some guidelines for RHC in patients with heart failure were provided by the 1995 report of the ACC/AHA Task Force on Practice Guidelines (Committee on Evaluation and Management of Heart Failure) (57). In some patients with dyspnea and/or pulmonary edema, a trial of diuretic and/or vasodilator therapy may be attempted without previous RHC; in others (e.g., some patients with normal left ventricular ejection fraction and suspected diastolic dysfunction), a strategy that would reduce preload may have deleterious consequences (58). Similarly, in some patients with shock, a therapeutic trial of volume infusion may be indicated; in others (especially those with respiratory distress who have not undergone endotracheal intubation and mechanical ventilation), such empiric therapy may also be associated with substantial risk. Right heart catheterization is warranted when these initial strategies are contraindicated or fail or when there are coexisting manifestations of "forward" (low output with hypotension, oliguria and/or azotemia) and "backward" (pulmonary venous hypertension with dyspnea and/or hypoxemia) heart failure. Similarly, RHC is indicated to determine the hemodynamic contribution to respiratory failure in patients with concurrent pulmonary and cardiac disease. In contrast, RHC is not warranted for the routine management of pulmonary edema, even if endotracheal intubation and mechanical ventilation have been necessary, or for the institution or titration of diuretic and vasodilator therapy in patients with mild or moderate heart failure.

RHC may be helpful to guide inotropic and pressor therapy in patients with severe decompensated heart failure and cardiogenic shock, respectively. It is warranted in the special cases of some patients with suspected pericardial tamponade, patients with decompensated heart failure undergoing noncardiac surgery and patients undergoing evaluation for heart transplantation. It should not be performed before pericardiocentesis in patients with hemodynamic instability when the diagnosis of pericardial tamponade is certain or probable. The use of RHC to guide diuretic and vasodilator therapy in patients with heart failure constitutes an intermediate indication over which there is substantial disagreement. Similarly, there is disagreement regarding the need for RHC in patients with compensated heart failure undergoing noncardiac surgery; use of RHC should be reserved for patients undergoing high risk operations.

Recommendations: Heart Failure

Conditions in which there is general agreement that RHC is warranted

1. Differentiation between hemodynamic and permeability pulmonary edema or dyspnea (or determination of the contribution of left heart failure to respiratory insufficiency in patients with concurrent cardiac and pulmonary disease) when a trial of diuretic and/or vasodilator therapy has failed or is associated with high risk.
2. Differentiation between cardiogenic and noncardiogenic shock when a trial of intravascular volume expansion has failed or is associated with high risk; guidance of pharmacologic and/or mechanical support.
3. Guidance of therapy in patients with concomitant manifestations of "forward" (hypotension, oliguria and/or azotemia) and "backward" (dyspnea and/or hypoxemia) heart failure.
4. Determination of whether pericardial tamponade is present when clinical assessment is inconclusive and echocardiography is unavailable, technically inadequate or nondiagnostic.
5. Guidance of perioperative management in selected patients with decompensated heart failure undergoing intermediate or high risk (3) noncardiac surgery.
6. Detection of the presence of pulmonary vasoconstriction and determination of its reversibility in patients being considered for heart transplantation.

Conditions in which reasonable differences of opinion exist regarding RHC

1. Differentiation between hemodynamic and permeability pulmonary edema or dyspnea (or determination of the contribution of left heart failure to respiratory insufficiency in patients with concurrent cardiac and pulmonary disease) when a trial of diuretic and/or vasodilator therapy is associated with low or intermediate risk.
2. Differentiation between cardiogenic and noncardiogenic shock when a trial of intravascular volume expansion is associated with intermediate risk.
3. Facilitation of titration of diuretic, vasodilator and inotropic therapy in patients with severe heart failure.
4. Guidance of perioperative management in patients with compensated heart failure undergoing intermediate or high risk (3) noncardiac surgery.

Conditions in which RHC is not warranted

1. Routine management of pulmonary edema, even if endotracheal intubation and mechanical ventilation have been necessary.
2. Differentiation between cardiogenic and noncardiogenic shock before a trial of intravascular volume expansion, when such a trial is associated with low risk.
3. Institution or titration of diuretic and/or vasodilator therapy in patients with mild or moderate heart failure.
4. Marked hemodynamic instability in patients in whom pericardial tamponade is certain or probable by clinical and/or echocardiographic criteria, and RHC would delay treatment.
5. Guidance of perioperative management in patients with compensated heart failure undergoing low risk (3) noncardiac surgery.

Conditions in which a relative contraindication to RHC exists

1. Coagulopathy (or anticoagulant therapy that cannot be temporarily discontinued).
2. Recent implantation of permanent pacemaker or cardioverter-defibrillator (in which case placement under fluoroscopic guidance is recommended).
3. Left bundle branch block (see "Acquisition and Interpretation of Data").
4. Bioprosthetic tricuspid (or pulmonic) valve.

Conditions in which an absolute contraindication to RHC exists

1. Right-sided endocarditis.
2. Mechanical tricuspid (or pulmonic) valve prosthesis.
3. Presence of thrombus or tumor in right heart chamber.
4. Terminal illness for which aggressive management is considered futile.

Acute Myocardial Infarction

Background. The routine use of RHC in uncomplicated acute myocardial infarction has never been recommended. Gore et al. (20) and Yarzebski et al. (59) examined the use of RHC over time in patients with acute myocardial infarction in Worcester, Massachusetts. They found an increase in the use of RHC from 1975 to 1984, followed by a progressive decline until 1990, the last year examined. Even before restrictions imposed by cost containment, managed care and published

guidelines, the cardiology community had already reduced the use of RHC in acute myocardial infarction on the basis of clinical judgment and experience.

Indications for the use of RHC are related to the diagnosis and management of *specific* complications of myocardial infarction. These include 1) hypotension, low cardiac output and cardiogenic shock as a consequence of predominant left ventricular failure; 2) acute mechanical complications (mitral regurgitation from papillary muscle rupture or ischemia, ventricular septal rupture or ventricular free wall rupture); 3) complicated right ventricular infarction; and 4) heart failure or pulmonary edema unresponsive to routine management.

The role of RHC has changed because of the availability of noninvasive diagnostic techniques, in particular, echocardiography. Echocardiography with Doppler imaging is the procedure of choice for the evaluation of left and right ventricular function and the diagnosis of complications of acute myocardial infarction (see later ["Alternative or Complementary Procedures"]). The major role of RHC is related to guiding the management of complications, such as therapy with vasoactive agents, intraaortic balloon pumping (IABP) and other interventions. If echocardiography with Doppler imaging is not available, RHC remains an important modality for the diagnosis of complications of myocardial infarction.

Hypotension, low cardiac output state and cardiogenic shock. RHC is most commonly used in the setting of acute myocardial infarction to guide the management of low cardiac output, hypotension and cardiogenic shock. Published guidelines consistently recommend the use of RHC in cardiogenic shock (5–7,10). Although the diagnosis of shock is dependent on the presence of hypotension and clinical signs of organ hypoperfusion (oliguria, cold skin, depressed mental status) (60), RHC is recommended to confirm the presence of shock by hemodynamic criteria and to assess filling pressures, thereby distinguishing cardiogenic from other forms of shock, in particular, hypovolemic shock. However, there are no randomized studies that address the benefit of RHC in this patient population. A nonrandomized study by Gore et al. (20) demonstrated excess mortality in all patients with acute myocardial infarction treated with RHC and no benefit in the subset of patients with cardiogenic shock. In that retrospective study, physician selection of the sicker patients to undergo RHC was likely. Similarly, Zion et al. (21) observed increased mortality in all patients with acute myocardial infarction managed with RHC but no difference in patients with cardiogenic shock. The study of Blumberg et al. (61), although not limited to patients with hypotension or cardiogenic shock, reported increased mortality in Medicare patients with acute myocardial infarction treated in hospitals with a higher rate of RHC.

The lack of observed benefit of RHC in the setting of cardiogenic shock may be in part related to the poor prognosis of these patients and the relative lack of effective treatment options, such as vasoactive agents, IABP (62) and thrombolytic agents (63) at the time these studies were carried out. Experimental and clinical studies suggest that aggressive use of IABP, often combined with thrombolysis, may improve prognosis (64,65). More definitive forms of coronary revascularization with percutaneous transluminal coronary angioplasty (PTCA) (66–68) or CABG (60,69–71) have revealed some encouraging results. The importance of the form of revascularization and the timing of the procedure in patients in cardiogenic shock are currently being studied by the international Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock (SHOCK) trial (60). Although a preliminary report from the registry of the SHOCK study (71) revealed a reduced mortality associated with the use of RHC in patients with suspected cardiogenic shock, there was substantial selection bias in that patients receiving RHC were younger and were also more likely to receive more aggressive management with IABP, cardiac catheterization, PTCA and CABG.

Acute mechanical complications. MITRAL REGURGITATION SECONDARY TO PAPILLARY MUSCLE RUPTURE. The presence of a flail mitral valve leaflet may be suspected on the basis of clinical presentation and physical examination, although in severe cases characterized by low cardiac output, the systolic murmur may not be heard because of rapid equalization of left ventricular and left atrial pressures (72,73). Acute mitral regurgitation can be accurately diagnosed by either transthoracic echocardiography or TEE; the latter is superior for detection of papillary muscle rupture (74). Severe mitral regurgitation associated with papillary muscle rupture usually prompts immediate cardiac catheterization (including RHC) to evaluate coronary anatomy before emergent surgical intervention. Bedside RHC is useful for guiding vasodilator, inotropic and intraaortic balloon therapy (75). On the basis of available published reports and experience, short-term RHC is useful for guiding therapy in patients who are not surgical candidates because of comorbidity or other reasons.

MITRAL REGURGITATION SECONDARY TO PAPILLARY MUSCLE ISCHEMIA. Papillary muscle dysfunction without mechanical disruption of the valve leads to mitral regurgitation, which may be transient. The clinical diagnosis may be confirmed by echocardiography with Doppler imaging. Treatment with antiischemic, diuretic and vasodilator agents can reduce ventricular size and alter ventricular geometry (76) and may diminish or eliminate mitral regurgitation. Bedside RHC is not required for diagnosis and may in fact be misleading. Although the presence of a "regurgitant" v wave on the PAOP tracing supports the diagnosis of mitral regurgitation, the height of the v wave often does not give an accurate assessment of the severity of the mitral regurgitation, and large v waves are not diagnostic of mitral regurgitation (77,78). Short-term hemodynamic monitoring is helpful in assessing therapeutic interventions, but the utility of RHC over clinical and echocardiographic assessment has not been studied in this setting.

VENTRICULAR SEPTAL RUPTURE. Echocardiography with Doppler imaging is highly sensitive and specific for the diagnosis of ventricular septal defect and provides information regarding the magnitude of the shunt, pulmonary artery pressure and right ventricular function. RHC, although useful for the diagnosis of postinfarction septal rupture (79), is in general not required. However, RHC is helpful for the assessment of

the magnitude of the shunt (when not measurable by echocardiography), evaluation of the response of the shunt to pharmacologic and IABP therapy and monitoring of patients in the postoperative period.

VENTRICULAR FREE WALL RUPTURE. Free wall rupture may present as (1) electromechanical dissociation; (2) subacute rupture with tamponade and a low output/shock state; or (3) pseudoaneurysm with or without hemodynamic instability. Rupture with electromechanical dissociation usually results in death before any attempt at premortem diagnosis. After subacute cardiac rupture, echocardiography with Doppler imaging is the primary modality for the diagnosis of pericardial effusion and tamponade and may guide pericardiocentesis. Echocardiography may also detect the presence of a pseudoaneurysm. RHC can confirm the diagnosis of pericardial tamponade in patients with suspected subacute cardiac rupture (80), in whom clinical, electrocardiographic (81) and echocardiographic features are not diagnostic. However, RHC should not delay potentially life-saving treatment.

Right ventricular infarction. Right ventricular dysfunction is a common complication of inferior wall myocardial infarction that can affect treatment and prognosis. The diagnosis of right ventricular infarction can be made by physical examination and electrocardiography, using right precordial leads (82); it can be confirmed by echocardiography or radionuclide imaging (83). Although several hemodynamic criteria have been proposed for the diagnosis of right ventricular infarction (84), RHC is usually not required for the diagnosis. Most patients with right ventricular infarction remain hemodynamically stable, although caution in the administration of nitrates and other vasodilators is recommended to avoid reduced filling of the impaired, volume-sensitive right ventricle (85). The degree of hemodynamic compromise depends on the extent of left and right ventricular as well as right atrial involvement (86,87). Furthermore, ventricular interaction through septal shift and pericardial restraint can markedly affect the systolic and diastolic function of both ventricles.

For patients with right ventricular infarction with hypotension and signs of low cardiac output, treatment includes cautious volume loading, low doses of inotropic agents and, if required, temporary (usually atrioventricular sequential) pacing (88). RHC is not required in the majority of these patients. Reperfusion of the infarct-related artery by either thrombolysis or catheter-based intervention can lead to rapid hemodynamic improvement (89,90). The degree of volume loading needed is controversial because excessive volume may result in a decline in cardiac output (88,91). Therefore, in patients with persistent hypotension or with low cardiac output despite initial treatment, RHC is indicated with particular attention to cardiac output measurements. Because of the alteration of the diastolic properties of the left ventricle caused by ventricular interaction and pericardial restraint, PAOP is not an accurate indicator of left ventricular preload (86). Careful monitoring of the effect of interventions on cardiac output is necessary.

Heart failure. RHC has not been shown to be effective for the diagnosis and guidance of management of congestive heart failure (CHF) in normotensive patients with acute myocardial infarction. Zion et al. (21) demonstrated increased mortality in patients with CHF receiving RHC, although there was no difference in mortality when the investigators controlled for severity of CHF. RHC in the patient with hypoxemia and myocardial ischemia may pose an increased risk. Use of RHC in patients with concurrent cardiac and pulmonary disease to determine the contribution of left heart failure to respiratory insufficiency and the use of RHC in patients with decompensated heart failure were discussed earlier under "Heart Failure."

Expert opinion of the Committee. Some guidelines for RHC in patients with acute myocardial infarction were provided in 1996 by the Report of the ACC/AHA Task Force on Practice Guidelines (Committee on Management of Acute Myocardial Infarction) (6).

In the setting of acute myocardial infarction with hemodynamic instability, the use of RHC depends on the underlying cause and the course of hemodynamic compromise. RHC is not required in patients with transient hypotension that responds promptly to either intravascular volume expansion or low doses of inotropic drugs, or both. In patients who do not improve rapidly, RHC is recommended to confirm the diagnosis of cardiogenic shock and to guide the assessment of the short-term response to inotropic, vasodilator and diuretic treatment or mechanical support (IABP). Similarly, transient hypotension in the setting of right ventricular infarction can often be managed without RHC. However, when hemodynamic compromise persists despite initial therapy, RHC is particularly important to monitor response of cardiac output to volume loading and other therapeutic measures.

In the setting of hemodynamic instability caused by structural complications after acute myocardial infarction, echocardiography with Doppler imaging is the primary procedure for diagnosis. The presence of acute mitral regurgitation with or without valve disruption, ventricular septal rupture or pericardial effusion or tamponade can be diagnosed rapidly by this modality. The major indications for RHC relate to hemodynamic monitoring of preoperative and postoperative pharmacologic interventions and mechanical (IABP) support.

In patients with heart failure or pulmonary edema (including those requiring mechanical ventilation) and in patients with right ventricular infarction *without persistent hypotension*, the use of RHC for the guidance of pharmacologic therapy represents an intermediate indication for which considerable disagreement exists. Furthermore, RHC over a longer period of time is controversial in patients with persistent hemodynamic instability who are not considered for coronary revascularization or surgical repair of structural complications.

Recommendations: Acute Myocardial Infarction

Conditions in which there is general agreement that RHC is warranted

1. Differentiation between cardiogenic and hypovolemic shock when initial therapy with intravascular volume expansion and low doses of inotropic drugs has failed.

2. Guidance of management of cardiogenic shock with pharmacologic and/or mechanical support in patients with and without coronary reperfusion therapy.
3. *Short-term* guidance of pharmacologic and/or mechanical management of acute mitral regurgitation (with or without disruption of the mitral valve) before surgical correction.
4. Establishment of severity of left to right shunting and short-term guidance of pharmacologic and/or mechanical management of ventricular septal rupture before surgical correction.
5. Guidance of management of right ventricular infarction with hypotension and/or signs of low cardiac output not responding to intravascular volume expansion, low doses of inotropic drugs and/or restoration of heart rate and atrioventricular synchrony.
6. Guidance of management of acute pulmonary edema not responding to treatment with diuretic drugs, nitroglycerin and other vasodilator agents and low doses of inotropic drugs.

Conditions in which reasonable differences of opinion exist regarding RHC

1. Guidance of ongoing management of hypotension responding to initial therapy with intravascular volume expansion and/or low doses of inotropic drugs.
2. *Short-term* guidance of pharmacologic and/or mechanical management of acute mitral regurgitation if operation is delayed or not contemplated.
3. Establishment of severity of left to right shunting, and *short-term* guidance of pharmacologic and/or mechanical management of ventricular septal rupture if operation is delayed or not contemplated.
4. Guidance of management of right ventricular infarction, after correction of hypotension and/or signs of low cardiac output by intravascular volume expansion, low doses of inotropic drugs and/or restoration of heart rate and atrioventricular synchrony.
5. Guidance of management of acute pulmonary edema with vasodilator agents and/or inotropic drugs after initial treatment with diuretic drugs and nitroglycerin has failed.
6. Confirmation of the diagnosis of pericardial tamponade associated with subacute myocardial rupture when clinical and echocardiographic assessments are inconclusive.

Conditions in which RHC is not warranted

1. Guidance of management of postinfarction angina.
2. Guidance of ongoing management of pulmonary edema responding promptly to treatment with diuretics and nitroglycerin.
3. Pericardial tamponade with marked hemodynamic instability, when the diagnosis is certain or likely by clinical and/or echocardiographic criteria, and RHC would delay treatment.

Conditions in which a relative contraindication to RHC exists

Thrombolytic and/or anticoagulation therapy (for additional relative contraindications, see "Heart Failure," above).

Conditions in which an absolute contraindication to RHC exists

See "Heart Failure," above.

Perioperative Use in Cardiac Surgery

Background. Patients undergoing cardiac surgery may develop disturbances of perfusion postoperatively that can be life threatening and/or place the function of vital organs at risk. Identification of the underlying hemodynamic derangements associated with impaired perfusion determines therapeutic strategy. RHC facilitates both the diagnosis and management of low cardiac output states, which may not always be adequately assessed by clinical markers of perfusion after cardiac surgery (92). Identification of abnormalities of cardiac output as well as right and left ventricular afterload or preload is particularly important to guide pharmacologic and other therapy aimed at optimizing perfusion. For example, information from RHC can be used to guide therapy with vasodilator, vasopressor and inotropic drugs. Finally, cardiac surgical techniques involving cardiopulmonary bypass may result in the accumulation of extravascular lung water, and RHC often serves as a useful guide for evaluating the effects of fluid management on global variables of perfusion after cardiac surgery.

Observational studies suggest that RHC is beneficial in cardiac surgical patients (93–96), although data collected from such patients have not definitively established improvement in outcome. Two large nonrandomized studies (97,98) have failed to detect any significant difference in outcome after CABG with RHC compared with central venous pressure monitoring alone. In another study (99), small numbers of patients initially randomized to undergo central venous pressure monitoring alone for cardiac surgery were crossed over to the RHC group because of the clinicians' opinion that it would be unethical to proceed without RHC in certain patients. The failure to demonstrate any difference in outcomes between RHC and central venous pressure monitoring may have been related to a small sample size, as well as bias caused by group reassignment.

The use of RHC in cardiac surgery with particular emphasis on CABG was recently discussed at the Pulmonary Artery Catheter Consensus Conference (7) and at the National Heart, Lung, and Blood Institute (NHLBI) and Food and Drug Administration (FDA) workshop on pulmonary artery catheterization and clinical outcomes (10). Both groups acknowledged that available data for use of RHC in the perioperative period are inconclusive because of methodologic difficulties. The Committee of the Pulmonary Artery Catheter Consensus Conference opined that the routine use of RHC does not appear to be necessary in *low risk* patients undergoing cardiac

surgery. They emphasized that RHC may be useful in high risk patients, particularly those with important left ventricular dysfunction, and proposed clinical studies in this patient category. The NHLBI and FDA workshop recommended a randomized, controlled trial to evaluate the need for and safety of RHC in low risk patients undergoing CABG.

Expert opinion of the Committee. In the absence of definitive prospective, randomized trials of RHC in patients undergoing cardiac surgery, the recommendations of the Committee are based on a consensus of expert opinion. Extensive clinical experience indicates that RHC is useful in patients undergoing cardiac surgery with previous evidence of ventricular dysfunction of any cause, complex coronary artery disease or valvular disease (especially when it is associated with significant coronary artery disease or pulmonary hypertension) and in patients requiring repeat cardiac surgery. Hemodynamic measurements obtained from RHC facilitate the management of patients after heart surgery and can be helpful in guiding inotropic, vasopressor, vasodilator and fluid therapy. RHC catheterization is warranted to facilitate the differentiation of hemodynamic abnormalities resulting in inadequate perfusion whenever therapeutic trials of intravascular volume expansion or vasoactive agents fail or when initial empiric management strategies are contraindicated because of inordinate risk (e.g., in patients with known severe ventricular dysfunction). The use of RHC after cardiac surgery should be discontinued when the benefit no longer outweighs the risks associated with catheter residence. This is especially important in the case of valvular surgery or heart transplantation, where infectious complications can have grave consequences.

Available data do not support the routine use of RHC after cardiac surgery in hemodynamically stable patients with good ventricular function. A rational alternative to routine RHC in this patient category is insertion of central venous access, which may be used as a conduit for RHC if hemodynamic inadequacy should become manifest. Justification for this approach includes the finding that no difference in measured outcomes could be detected in a controlled observational study of cardiac surgical patients who developed a hemodynamic "need" for RHC after initial monitoring with central venous cannulation alone (98). Alternative methods for the evaluation of cardiac function, such as echocardiography, should also be considered, although continual monitoring of echocardiographic variables in the postoperative period is not practical or feasible with currently available technology (see "Alternatives or Complementary Procedures," below).

Recommendations: Perioperative Use in Cardiac Surgery

Conditions in which there is general agreement that RHC is warranted

1. Differentiation between causes of low cardiac output (hypovolemia vs. ventricular dysfunction), when clinical and/or echocardiographic assessment is inconclusive.
2. Differentiation between right and left ventricular dysfunction and pericardial tamponade, when clinical and/or echocardiographic assessment is inconclusive.
3. Guidance of management of severe low cardiac output syndromes.
4. Diagnosis and guidance of management of pulmonary hypertension in patients with systemic hypotension and evidence of inadequate organ perfusion.

Conditions in which reasonable differences of opinion exist regarding RHC

1. Guidance of inotropic and/or vasopressor therapy after patients with significant cardiac dysfunction have achieved hemodynamic stability.
2. Guidance of management of hypotension and evidence of inadequate organ perfusion when a therapeutic trial of intravascular volume expansion and/or vasoactive agents is associated with moderate risk.

Conditions in which RHC is not warranted

1. Routine management of uncomplicated cardiac surgical patients with good ventricular function and hemodynamic stability.
2. Initial management of postoperative hypotension when a therapeutic trial of volume expansion and/or vasoactive agents is associated with low risk.

Conditions in which a relative or absolute contraindication to RHC exists

See "Heart Failure," above.

The role of RHC in the perioperative management of high risk cardiac patients after *noncardiac* surgery is not discussed in this Expert Consensus Document, with the exception of heart failure (see "Heart Failure," above). These issues have been addressed by the practice guidelines of the ACC/AHA on perioperative cardiovascular evaluation for noncardiac surgery (3) and by the practice guidelines of the American Society of Anesthesiologists on pulmonary artery catheterization (8).

Primary Pulmonary Hypertension

Background. Primary pulmonary hypertension (PPH) is a life-threatening disease of unknown etiology that is characterized by persistent and usually progressive elevation of pulmonary artery pressure and pulmonary vascular resistance. The recent data from a multicenter registry (100) organized by the National Institutes of Health (NIH) have shown that the median survival after diagnosis is <3 years, although rare individual examples of spontaneous remission and prolonged survival have been reported (101,102).

Although the clinical value of RHC in patients with suspected or confirmed PPH has not been formally assessed by randomized prospective clinical trials, RHC has been exten-

sively used in such patients for (1) establishment of the diagnosis and severity of pulmonary hypertension; (2) assessment of prognosis based on hemodynamic parameters; and (3) selection and assessment of response to pharmacologic agents (100–105).

Diagnostic applications. For accurate assessment of the severity of pulmonary hypertension and for establishing the diagnosis of precapillary pulmonary hypertension, RHC is required. Doppler echocardiography can be used to estimate pulmonary artery systolic pressure in patients with at least mild tricuspid regurgitation. However, measurement of PAOP, which is essential to distinguish between postcapillary (elevated PAOP) and precapillary (normal PAOP) pulmonary hypertension and to determine pulmonary vascular resistance, cannot presently be accomplished without RHC. Several studies (100–102) have shown that the outcome in PPH is closely related to the severity of hemodynamic derangements identified by RHC. Hemodynamic variables predictive of poor survival include mean pulmonary artery pressure >85 mm Hg, right atrial pressure >20 mm Hg, cardiac index <2.0 liters/min per m^2 and mixed venous oxygen saturation <60% (100–103).

Therapeutic applications. Before the institution of long-term pharmacologic therapy in PPH, targeted to produce pulmonary vasodilation, the hemodynamic response to vasodilator agents is usually determined by RHC. Several studies (101,106–120a) have correlated the pattern of acute hemodynamic response with long-term prognosis and the clinical response to long-term vasodilator therapy.

The optimal response to vasodilator therapy is generally considered to be a decrease in pulmonary artery pressure and pulmonary vascular resistance and an increase in cardiac output, without hypoxemia, tachycardia or an excessive decrease in systemic arterial pressure. Long-term vasodilator therapy is also considered when there is a decrease only in pulmonary vascular resistance, without a major decrease in pulmonary artery pressure. A substantial (20% to 30%) reduction in pulmonary vascular resistance in response to acute vasodilator therapy has been shown to predict an improved outcome (109,115,116). Adverse hemodynamic responses include reduction of cardiac output and systemic arterial pressure and an increase in right atrial pressure, suggesting right ventricular failure. Such responses are regarded as contraindications to long-term vasodilator therapy. Responders and nonresponders can be identified *only* by careful hemodynamic evaluation with RHC. It is particularly important to emphasize that empiric treatment of patients with PPH with vasodilator agents, including oral calcium channel blockers, can be hazardous (113). However, it has been observed (118) that the lack of a beneficial hemodynamic response to acute intravenous administration of prostacyclin does not necessarily imply lack of favorable response during long-term therapy.

Technical considerations. Performance of RHC in patients with pulmonary hypertension may be difficult, particularly in patients with right heart dilation and tricuspid regurgitation. Fluoroscopic guidance of RHC may be useful. An increased risk of pulmonary artery rupture, related to inflation of the catheter tip balloon when advanced too far distally, has been observed. Therefore, placement of the catheter tip in the proximal pulmonary artery has been recommended. Use of a guide wire balloon flotation catheter facilitates its placement and reduces the frequency of dislodgment from the pulmonary artery.

Recommendations: PPH

Conditions in which there is general agreement that RHC is warranted

1. Exclusion of postcapillary (elevated PAOP) causes of pulmonary hypertension.
2. Establishment of diagnosis and assessment of severity of precapillary (normal PAOP) pulmonary hypertension.
3. Selection and establishment of safety and efficacy of long-term vasodilator therapy based on acute hemodynamic response.
4. Assessment of hemodynamic variables before lung transplantation.

Conditions in which reasonable differences of opinion exist regarding RHC

1. Evaluation of long-term efficacy of vasodilator therapy, particularly prostacyclin.
2. Exclusion of significant left to right or right to left intracardiac shunt.

Conditions in which RHC is not warranted

None.

Conditions in which a relative or absolute contraindication to RHC exists

See “Heart Failure,”above.

Complications

Complications related to RHC are associated with the establishment of central venous access, the catheterization procedure itself and catheter residence. Adverse events related to central venous cannulation include arterial puncture, bleeding at the site of insertion, nerve injury, pneumothorax and air embolism. These events may occur with any central venous cannulation and are not unique to RHC (121).

The primary complications related to the RHC procedure itself are arrhythmias, which are usually minor in nature (premature ventricular or atrial contractions) and resolve spontaneously after the catheter is advanced through the right heart into the pulmonary artery. Clinically insignificant ventricular arrhythmias, usually not requiring therapy, have been reported to occur in ~30% to 60% of patients during RHC (121,122). Sustained ventricular arrhythmias requiring treatment occur much less commonly (121,123–125) and primarily

occur in patients with myocardial ischemia or myocardial infarction or preexisting ventricular arrhythmias. RHC can also occasionally precipitate right bundle branch block; in patients with preexisting left bundle branch block, this can result in complete heart block (126). On rare occasions, knotting of the catheter can occur during insertion (127), emphasizing the importance of fluoroscopic evaluation of the catheter position after the procedure.

Arguably, the most serious complications of RHC are related to catheter residence. They include pulmonary artery rupture (128–131), thrombophlebitis (132), venous or intracardiac thrombus formation (132,133), pulmonary infarction (134,135), endocarditis (136,137) and other catheter-related infections (121,136,138). Risk factors for pulmonary artery rupture include pulmonary hypertension and recent cardiopulmonary bypass (139,140). The incidence of pulmonary infarction of embolic, thrombotic or ischemic origin has been reported to be 0% to 1.3% (134,135). The risk of thrombotic and infectious complications increases with duration of catheter residence (136). Most data suggest that the risk of infection increases significantly when catheters remain in place >3 to 4 days (121,141). Local infection (catheter colonization) has been observed in 18% to 63% of patients with catheters in place for an average of 3 days, whereas bloodstream infection has been documented in up to 5% of cases with a similar duration of catheterization (141).

Alternative or Complementary Procedures

Echocardiography with Doppler imaging is the most important alternative or complementary procedure to RHC in the critically ill patient. Although echocardiography has not been validated for widespread clinical use as an alternative to RHC for estimation of left heart filling pressure, stroke volume and cardiac output, it provides a wealth of complementary information regarding global and regional abnormalities of left and right ventricular function, cardiac chamber size, valvular lesions, ventricular wall thickness and pericardial structure and function, in particular, the presence and physiologic significance of pericardial effusion. In the severely ill patient, TTE may be limited by lack of cooperation of the patient, mechanical ventilation and other factors, in which case TEE is superior. When performed with careful sedation and close monitoring, TEE has a very low complication rate, even among the most seriously ill patients (142–144), when used with appropriate caution.

Echocardiographic acquisition of anatomic and physiologic data. *Hypotension and shock.* Echocardiography can elucidate the likely mechanism of hypotension and shock (143,145). Assessment of ventricular and valvular function aids in the differentiation of patients with cardiogenic from those with noncardiogenic shock (146). The presence and hemodynamic significance of pericardial effusion can be determined by the assessment of right ventricular wall motion and variation of transmitral flow (147,148).

Mechanical complications of myocardial infarction. MITRAL REGURGITATION. TTE is 100% sensitive for the detection of mitral regurgitation resulting from papillary muscle rupture, infarction or ischemia and in distinguishing it from ventricular septal defect (149). The degree of mitral regurgitation and the effectiveness of therapy can be evaluated by echocardiography (150).

VENTRICULAR SEPTAL RUPTURE. Ventricular septal rupture can be detected by TTE and Doppler imaging with a sensitivity >90% (74,149,151). Helmcke et al. (152) demonstrated at cardiac surgery a correlation between the width of the jet and the size of the defect. When TTE imaging is suboptimal, TEE is highly accurate (74). In the absence of valvular regurgitation, the degree of left to right shunting can be estimated by Doppler calculation of right and left ventricular stroke volumes (153).

VENTRICULAR FREE WALL RUPTURE. Ventricular free wall rupture, which may be associated with pericardial tamponade, can be suspected or diagnosed by echocardiography. Thrombus in the pericardial space supports the diagnosis. Echocardiography has a high sensitivity for the diagnosis of the subacute form of cardiac rupture, revealing findings of (sometimes localized) pericardial effusion and associated tamponade (154). A pseudoaneurysm, resulting from subacute rupture, can be suggested by echocardiography (155), although it may be difficult to differentiate from a true aneurysm (156). Doppler flow imaging demonstrates a characteristic flow pattern in and out of the pericardial cavity at the site of the tear (151,157).

Right ventricular infarction. Echocardiography supports the diagnosis of right ventricular infarction (158). Abnormal right ventricular wall motion in the presence of an inferoposterior myocardial infarction is diagnostic of this event. The presence of normal or only mildly impaired left ventricular systolic function is suggestive of the need for intravascular volume expansion as initial therapy. In the setting of a large right ventricular infarction with right ventricular failure, a right to left shunt across a patent foramen ovale can be detected by Doppler imaging (159).

Doppler estimation of cardiac pressures and stroke volume. *Pulmonary artery pressure.* Several studies (160–162) have demonstrated the utility of Doppler echocardiography to obtain hemodynamic information in patients with cardiac disease. In patients with at least a minor degree of tricuspid regurgitation, the difference between right ventricular systolic pressure and right atrial pressure may be estimated by Doppler echocardiography from the simplified Bernoulli equation (163). Right atrial pressure may be estimated from clinical examination (or measured by central venous pressure monitoring) or assigned an "average" value such as 10 mm Hg (164). Pulmonary artery systolic pressure can thus be estimated as the sum of the transtricuspid gradient and right atrial pressure because in the absence of pulmonary outflow obstruction, pulmonary artery and right ventricular systolic pressures are equal.

Left ventricular filling pressure. Considerable effort has been directed at estimating left ventricular filling pressure, assessed by Doppler ultrasound from the pattern of left ventricular

filling (165,166). The peak rate of early diastolic filling of the ventricle (E), the E wave deceleration time, the ratio of early to late filling (E/A), the isovolumic relaxation time (IVRT) and the atrial filling fraction are all related to the filling pressure but are also affected by the intrinsic diastolic properties of the ventricle (16). Rossvoll and Hatle (167) demonstrated that an elevated left ventricular filling pressure (PAOP >15 mm Hg) could be predicted with 85% sensitivity by examining both the pulmonary venous and transmitral A waves. None of these techniques for noninvasive estimation of left ventricular filling pressure are used routinely, and they remain the subject of continued clinical investigation.

Stroke volume. Doppler echocardiography can be used to calculate stroke volume. Calculation is accomplished by integrating the area under the flow velocity integral across any of the four cardiac valves and multiplying this value by the cross-sectional area of the annulus at which the measurement is obtained. Although not widely applied to critically ill patients, these measurements have been shown (168,169) to be reproducible and to correlate well with measurements obtained by RHC. Application of these principles allows for calculation of the regurgitant fraction (170) and the magnitude of intracardiac shunts (Qp/Qs) (171). However, more experience is needed before these techniques can be applied in widespread clinical practice.

TEE in the perioperative period. TEE is used extensively before, during and after cardiac surgery (172–174). TEE plays an important role in evaluating left ventricular function and the results of valve repair. Echocardiographic assessment is of particular value in the patient with hypotension and low cardiac output during separation from cardiopulmonary bypass. In this setting, TEE can be used to determine the likely cause of hypotension, differentiating among causes such as abnormal left ventricular systolic function, mitral insufficiency and an underfilled left ventricle.

A variety of promising developments, including miniaturized probes and techniques for real time on-line monitoring of ventricular volumes, ejection fraction and compliance, may further enhance the role of cardiac ultrasound in the perioperative period. Although of promise, these techniques have not been validated for clinical use.

Cost-Effectiveness of RHC

Cost-effectiveness analysis can be used to compare competing forms of therapy or courses of medical care and can offer a cost-effectiveness ratio, best expressed in cost per quality-adjusted life-year gained (175). This ratio for a specific intervention may then be compared with ratios for other interventions competing for scarce health care resources. To perform such analysis, it is necessary to measure both the effect and cost of the medical intervention and then extrapolate short-term results over a lifetime, to derive the cost per quality-adjusted life-year gained. In the case of RHC, to establish truly comparable groups that would provide usable cost and efficacy data, it would be necessary to conduct randomized trials. However, as noted below, such trials may be difficult to conduct successfully in homogeneous populations.

Given these difficulties, there is limited information permitting evaluation of the cost-effectiveness of RHC. If, in some patients, there is clinical benefit at reduced cost, then RHC is clearly indicated. In patients in whom there is decreased or negative benefit, the procedure is not indicated regardless of cost. In patients in whom there is benefit at increased cost, a formal cost-effectiveness analysis may help in deciding whether to place a right heart catheter. At least one attempt to provide a theoretic evaluation of the cost-effectiveness of RHC in patients undergoing CABG has been published (176). Further research may help to define the cost-effectiveness of RHC in different patient groups, especially those in which the value of RHC is currently controversial.

Proposals for Randomized Trials

The Expert Consensus Committee considered carefully the role of future research for evaluation of the safety and efficacy of RHC in patients with cardiac disease. It identified two areas in which RHC is frequently used and randomized trials may be considered, namely, heart failure and cardiac surgery. The Committee came to the following conclusions:

Randomized trials in heart failure. Randomized clinical trials in patients with heart failure are ethical in those subsets (e.g., patients with severe heart failure who require titration of diuretic, vasodilator and inotropic therapy) for which reasonable differences of opinion exist regarding the indication for RHC. Spodick (177) has suggested that a panel of three experts be formed in each participating hospital; randomization would be permissible only if there were a disagreement among the three experts as to whether RHC was indicated in an individual patient. Although such an approach would be difficult, it would facilitate and support recruitment of appropriate patients for such a trial. For consideration of sample size and power, end points other than mortality would be necessary. End points should also include morbidity, length of ICU and total hospital stay and cost. Optimal use of RHC should be examined, with attention to 1) appropriateness of indications; 2) skill of personnel; 3) accuracy of measurements; 4) skill in interpretation of measurements; and 5) management algorithms based directly on RHC measurements. For the benefit of RHC to be assessed, it must be deployed in clinical conditions for which beneficial treatment is available. Patients should be stratified according to whether heart failure is acute or chronic and whether shock is present.

A multicenter trial on the effectiveness of RHC in patients with persistent or refractory CHF has been proposed at the recent NHLBI/FDA Workshop on Pulmonary Artery Catheterization and Clinical Outcomes (10).

Randomized trials in cardiac surgery. RHC is commonly performed as a routine part of CABG and other cardiac surgery. At present, the routine use of RHC for *low risk* patients (normal ventricular function and hemodynamically stable) cannot be viewed as warranted. In principle, the routine

use of RHC could be established after an appropriate clinical trial. However, such a trial would be difficult to perform because the event rates are low in this patient category, and the impact of RHC is likely to be small. In low risk patients, for whom the mortality rate may be ≤1%, RHC would be expected to have minimal benefit and might add risk. RHC is even less likely to favorably affect other potential end points, such as infection, stroke and myocardial infarction. Cost and resource use could be considered an end point, but in the absence of sufficient power for examining a clinical end point, a cost-effectiveness study could not be reasonably performed. However, if clinical outcome is considered likely to be equivalent with or without the use of RHC, then a pure cost study (i.e., a cost minimization study) would be possible.

A clinical trial in *high risk* patients undergoing cardiac surgery is potentially more likely to be revealing because the incidence of end points will be much higher, and the potential of RHC to affect outcome would appear to be much greater than a trial in low risk patients. Clinical studies in this patient category have been recently proposed by the Pulmonary Artery Catheter Consensus Conference (7). However, the Expert Consensus Committee of this document is concerned that such a trial is difficult to perform because the clinical situation is not in equipoise. Anesthesiologists and cardiac surgeons in this situation are likely to regard RHC as useful, making patient enrollment difficult. Should such a trial be performed, a reasonable primary clinical end point will need to be identified, with cost and resources use as secondary end points. Because mortality is not likely to vary enough between treatment arms to permit adequate statistical power at a reasonable sample size, it will be necessary to nominate a different clinical end point or composite of end points.

Summary. Although it is possible in principle to perform clinical trials in patients with heart failure undergoing cardiac surgery, the obstacles are considerable. Randomized trials most commonly assess therapeutic interventions. RHC, in contrast, is a diagnostic technique that may result in multiple changes in therapeutic strategies, from relatively minor (e.g., changing diuretic agents) to major (e.g., a decision on the need for cardiac surgery of even transplantation). Sample sizes supporting the selection of mortality as the primary end point would be unreasonably large. Given the difficulties noted above, the most compelling case can probably be made for a trial involving the role of RHC in the management of refractory decompensated heart failure.

Frequency of Use of RHC

There are presently no good data available regarding the frequency of use of bedside RHC in cardiac practice. Data provided by industry revealed an annual sale of ~1.5 million (Swan-Ganz) catheters in the United States, with estimates that 30% of catheters are used in cardiac surgery, 30% in cardiac catheterization laboratories and coronary care units, 25% in high risk surgery and trauma and 15% in medical ICUs (George Sopko, MD, NHLBI, personal communication, 1997 [to B. Bernard, Baxter Health Care Corporation]). A question on the frequency of use of Swan-Ganz catheters was included in the 1997 ACC Membership Profile. This survey is mailed biannually to all ACC members to ascertain selected member attributes, including practice characteristics and educational interests. The 1997 Membership Profile achieved a response rate of 59% (11,502 questionnaires returned out of 19,421 mailed). Respondents were asked to indicate how many times a year they performed Swan-Ganz catheterization in patients with cardiac disease (excluding use in the medical ICU and surgery). The question was answered by 6,985 respondents. Five frequency of use categories were available: category 1 = None; category 2 = 1 to 50; category 3 = 51 to 100; category 4 = 101 to 150; and category 5 = more than 150. Most respondents (65%) indicated that they performed between 1 and 50 Swan-Ganz catheterizations/year. Respondents self-identified as either invasive or interventional cardiologists reported the highest frequency of Swan-Ganz catheterizations in the 1 to 50 range (72% vs. 61% for all other respondents). The differential in frequency of use between this group of invasive or interventional cardiologists and other cardiovascular practitioners was greater in the higher frequency of use categories (26% vs. 9% for procedure frequencies >50/year). These data cannot address whether use in the 1 to 50 category is more skewed to the lower or higher part of this range.

Conclusions

The Expert Consensus Committee was convened to formulate recommendations for the use of bedside RHC in patients with known or suspected cardiac disease. Because few data are available from well controlled clinical trials, the recommendations are based primarily on inferences from published reports and expert opinion. The use of RHC over the past quarter century has fostered major advances in the diagnosis and treatment of critically ill cardiac patients. The role of RHC is currently in transition because of the availability of noninvasive diagnostic techniques, in particular, echocardiography with Doppler imaging, which in some cases complements and in others is an alternative to RHC. RHC has a primary role in management of heart failure in certain patients in whom 1) the diagnosis of heart failure is uncertain; 2) heart failure is of sufficient severity to require intensive pharmacologic management; or 3) heart transplantation is contemplated. In patients with acute myocardial infarction, echocardiography has a primary role in the diagnosis of mechanical complications, and RHC is helpful in certain instances for assessment of the severity of hemodynamic compromise and the response to therapy. The Committee does not recommend the routine use of RHC in uncomplicated cardiac surgical patients with good ventricular function and hemodynamic stability.

Although the Committee recognizes the interest in randomized clinical trials for establishing the safety and efficacy of RHC, it is not optimistic that such studies will be feasible. The challenge of controlling for the effects of therapeutic interventions based on the diagnostic modality of RHC while achieving

adequate statistical power in well defined patient subgroups will be difficult to meet. A possible exception would be a study of the efficacy of RHC in the management of patients with refractory decompensated heart failure. The Committee questions whether major resources should be devoted to reevaluation of the role of RHC or whether such resources would be better directed toward further development and evaluation of semi-invasive or noninvasive diagnostic techniques, including echocardiography with Doppler imaging.

We thank Lori Fields and Carmen L. Vera for preparation of the manuscript and Joseph Cosico, RN, MA, CCRN for administrative assistance. We also thank the members of the ACC staff for their support.

Staff, American College of Cardiology

Christine McEntee, Executive Vice President
David P. Bodycombe, ScD, Director, Research and Information Management
Reneé L. Hemsley, Research and Information Management
Dionna D. Green, Research and Information Management

References

1. Connors AF Jr, Speroff T, Dawson NV, et al. The effectiveness of right heart catheterization in the initial care of critically ill patients (SUPPORT Investigators). JAMA 1996;276:889–97.
2. Dalen JE, Bone RC. Is it time to pull the pulmonary artery catheter? JAMA 1996;276:916–8.
3. Eagle KA, Brundage BH, Chaitman BR, et al. Guidelines for perioperative cardiovascular evaluation for noncardiac surgery: report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Perioperative Cardiovascular Evaluation for Noncardiac Surgery). J Am Coll Cardiol 1996;27:910–48.
4. Swan HJ, Ganz W, Forrester J, et al. Catheterization of the heart in man with use of a flow-directed balloon-tipped catheter. N Engl J Med 1970;283:447–51.
5. Friesinger II GC, Williams SV. Clinical competence in hemodynamic monitoring: a statement for physicians from the ACP/ACC/AHA Task Force on Clinical Privileges in Cardiology. J Am Coll Cardiol 1990;15: 1460–4.
6. Ryan TJ, Anderson JL, Antman EM, et al. ACC/AHA guidelines for the management of patients with acute myocardial infarction: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Management of Acute Myocardial Infarction). J Am Coll Cardiol 1996;28:1328–428.
7. Pulmonary Artery Catheter Concensus Conference Participants. Pulmonary Artery Catheter Consensus Conference: consensus statement. Crit Care Med 1997;25:910–25.
8. American Society of Anesthesiologists Task Force on Pulmonary Artery Catheterization. Practice guidelines for pulmonary artery catheterization: a report by the American Society of Anesthesiologists Task Force on Pulmonary Artery Catheterization. Anesthesiology 1993;78:380–94.
9. Naylor CD, Sibbald WJ, Sprung CL, et al. Pulmonary artery catheterization: can there be an integrated strategy for guideline development and research promotion? JAMA 1993;269:2407–11.
10. Sopko G, Gordon B, Wood S. Pulmonary Artery Catheterization and Outcomes: NHLBI and FDA workshop. JAMA. Submitted for publication.
11. Iberti TJ, Fischer EP, Leibowitz AB, et al. A multicenter study of physicians' knowledge of the pulmonary artery catheter: Pulmonary Artery Catheter Study Group. JAMA 1990;264:2928–32.
12. Gnaegi A, Feihl F, Perret C. Intensive care physicians' insufficient knowledge of right-heart catheterization at the bedside: time to act? Crit Care Med 1997;25:213–20.
13. Iberti TJ, Daily EK, Leibowitz AB, et al. Assessment of critical care nurses' knowledge of the pulmonary artery catheter: the Pulmonary Artery Catheter Study Group. Crit Care Med 1994;22:1674–8.
14. Foster E, Schiller NB. Transesophageal echocardiography in the critical care patient. Cardiol Clin 1993;11:489–503.
15. Kuecherer HF, Muhiudeen IA, Kusumoto FM, et al. Estimation of mean left atrial pressure from transesophageal pulsed Doppler echocardiography of pulmonary venous flow. Circulation 1990;82:1127–39.
16. Nagueh SF, Kopelen HA, Zoghbi WA. Feasibility and accuracy of Doppler echocardiographic estimation of pulmonary artery occlusive pressure in the intensive care unit. Am J Cardiol 1995;75:1256–62.
17. Poelaert JI, Trouerbach J, De Buyzere M, et al. Evaluation of transesophageal echocardiography as a diagnostic and therapeutic aid in a critical care setting. Chest 1995;107:774–9.
18. Spencer KP, Krauss D, Thurn J, et al. Transnasal transesophageal echocardiography. J Am Soc Echocardiogr 1997;10:728–37.
19. Baxter Jr, JK, Beilman GJ. Effectiveness of right heart catheterization: time for a randomized trial [letter]. JAMA 1997;277:108.
20. Gore JM, Goldberg RJ, Spodick DH, et al. A community-wide assessment of the use of pulmonary artery catheters in patients with acute myocardial infarction. Chest 1987;92:721–7.
21. Zion MM, Balkin J, Rosenmann D, et al. Use of pulmonary artery catheters in patients with acute myocardial infarction: analysis of experience in 5,841 patients in the SPRINT Registry (SPRINT Study Group). Chest 1990;98: 1331–5.
22. Lin DY, Psaty BM, Kronmal RA. Assessing the sensitivity of regression results to unmeasured confounders in observational studies. Biometrics 1998;54:948–63.
23. Freedman B. Equipoise and the ethics of clinical research. N Engl J Med 1987;317:141–5.
24. Guyatt G. A randomized control trial of right-heart catheterization in critically ill patients: Ontario Intensive Care Study Group. J Intensive Care Med 1991;6:91–5.
25. Cook D, Randolph A, Kernerman P, et al. Central venous catheter replacement strategies: a systematic review of the literature. Crit Care Med 1997;25:1417–24.
26. Courtois M, Fattal PG, Kovács Jr, SJ, et al. Anatomically and physiologically based reference level for measurement of intracardiac pressures. Circulation 1995;92:1994–2000.
27. Carey CF, Courtois M, Barzilai B, et al. Correct zero reference leveling for the pulmonary artery catheter [abstract]. Circulation 1995;94 Suppl I:387–8.
28. West J, Dollery CT, Naimark A, et al. Distribution of blood flow in isolated lung: relation to vascular and alveolar pressures. J Appl Physiol 1964;19: 713–24.
29. Ermakov S, Hoyt JW. Pulmonary artery catheterization. Crit Care Clin 1992;8:773–806.
30. Cope DK, Grimbert F, Downey JM, et al. Pulmonary capillary pressure: a review. Crit Care Med 1992;20:1043–56.
31. Rajacich N, Burchard KW, Hasan FM, et al. Central venous pressure and pulmonary capillary wedge pressure as estimates of left atrial pressure: effects of positive end-expiratory pressure and catheter tip malposition. Crit Care Med 1989;17:7–11.
32. Shasby DM, Dauber IM, Pfister S, et al. Swan-Ganz catheter location and left atrial pressure determine the accuracy of the wedge pressure when positive end-expiratory pressure is used. Chest 1981;80:666–70.
33. Lozman J, Powers Jr, SR, Older T, et al. Correlation of pulmonary wedge and left atrial pressures: a study in the patient receiving positive end expiratory pressure ventilation. Arch Surg 1974;109:270–7.
34. Gershan JA. Effect of positive end-expiratory pressure on pulmonary capillary wedge pressure. Heart Lung 1983;12:143–8.
35. Bridges EJ, Woods SL. Pulmonary artery pressure measurement: state of the art. Heart Lung 1993;22:99–111.
36. Levy MM. Pulmonary capillary pressure: clinical implications. Crit Care Clin 1996;12:819–39.
37. Crexells C, Chatterjee K, Forrester JS, et al. Optimal level of filling pressure in the left side of the heart in acute myocardial infarction. N Engl J Med 1973;289:1263–6.
38. Stevenson LW, Tillisch JH. Maintenance of cardiac output with normal filling pressures in patients with dilated heart failure. Circulation 1986;74: 1303–8.
39. Cigarroa RG, Lange RA, Williams RH, et al. Underestimation of cardiac

output by thermodilution in patients with tricuspid regurgitation. Am J Med 1989;86:417–20.
40. Chatterjee K, Swan HJ, Ganz W, et al. Use of a balloon-tipped flotation electrode catheter for cardiac monitoring. Am J Cardiol 1975;36:56–61.
41. Vincent JL, Thirion VM, Brimioulle P, et al. Thermodilution measurement of right ventricular ejection fraction. Intensive Care Med 1986;12:33–8.
42. Normann RA, Johnson RW, Messinger JE, et al. A continuous cardiac output computer based on thermodilution principles. Ann Biomed Eng 1989;17:61–73.
43. Segal J, Pearl RG, Ford Jr, AJ, et al. Instantaneous and continuous cardiac output obtained with a Doppler pulmonary artery catheter. J Am Coll Cardiol 1989;13:1382–92.
44. Stevenson LW, Perloff JK. The limited reliability of physical signs for estimating hemodynamics in chronic heart failure. JAMA 1989;261:884–8.
45. Dash H, Lipton MJ, Chatterjee K, et al. Estimation of pulmonary artery wedge pressure from chest radiograph in patients with chronic congestive cardiomyopathy. Br Heart J 1980;12:143–8.
46. Fein AM, Goldberg SK, Walkenstein MD, et al. Is pulmonary artery catheterization necessary for the diagnosis of pulmonary edema? Am Rev Respir Dis 1984;129:1006–9.
47. Connors AF Jr, McCaffree DR, Gray BA. Evaluation of right-heart catheterization in the critically ill patient without acute myocardial infarction. N Engl J Med 1983;308:263–7.
48. Mimoz O, Rauss A, Rekik N, et al. Pulmonary artery catheterization in critically ill patients: a prospective analysis of outcome changes associated with catheter-prompted changes in therapy. Crit Care Med 1994;22:573–9.
49. Hancock EW. Subacute effusive-constrictive pericarditis. Circulation 1971; 43:183–92.
50. Matthay MA, Chatterjee K. Bedside catheterization of the pulmonary artery: risks compared with benefits. Ann Intern Med 1988;109:826–34.
51. Stevenson LW, Dracup KA, Tillisch JH. Efficacy of medical therapy tailored for severe congestive heart failure in patients transferred for urgent cardiac transplantation. Am J Cardiol 1989;63:461–4.
52. Stevenson LW, Tillisch JH, Hamilton M, et al. Importance of hemodynamic response to therapy in predicting survival with ejection fraction less than or equal to 20% secondary to ischemic or nonischemic dilated cardiomyopathy. Am J Cardiol 1990;66:1348–54.
53. Steimle AE, Stevenson LW, Chelimsky-Fallick C, et al. Sustained hemodynamic efficacy of therapy tailored to reduce filling pressures in survivors with advanced heart failure. Circulation 1997;96:1165–72.
54. Addonizio LJ, Gersony WM, Robbins RC, et al. Elevated pulmonary vascular resistance and cardiac transplantation. Circulation 1987;76:52–5.
55. Costard-Jackle A, Fowler MB. Influence of preoperative pulmonary artery pressure on mortality after heart transplantation: testing of potential reversibility of pulmonary hypertension with nitroprusside is useful in defining a high risk group. J Am Coll Cardiol 1992;19:48–54.
56. Semigran MJ, Cockrill BA, Kacmarek R, et al. Hemodynamic effects of inhaled nitric oxide in heart failure. J Am Coll Cardiol 1994;24:982–8.
57. Williams JF, Bristow MR, Fowler MB, et al. Guidelines for the evaluation and management of heart failure. Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Evaluation and Management of Heart Failure). J Am Coll Cardiol 1995;26:1376–98.
58. Goldsmith SR, Dick C. Differentiating systolic from diastolic heart failure: pathophysiologic and therapeutic considerations. Am J Med 1993;95:645–55.
59. Yarzebski J, Goldberg RJ, Gore JM, et al. Temporal trends and factors associated with pulmonary artery catheterization in patients with acute myocardial infarction. Chest 1994;105:1003–8.
60. Hochman JS, Boland J, Porway M, et al. Current spectrum of cardiogenic shock and effect of early revascularization on mortality: results of an international registry. (SHOCK Registry Investigators). Circulation 1995; 91:873–81.
61. Blumberg MS, Binns GS. Swan-Ganz catheter use and mortality of myocardial infarction patients. Health Care Financing Rev 1994;15:91–103.
62. DeWood MA, Notske RN, Hensley GR, et al. Intraaortic balloon counterpulsation with and without reperfusion for myocardial infarction shock. Circulation 1980;61:1105–12.
63. Califf RM, Bengtson JR. Cardiogenic shock. N Engl J Med 1994;330:1724–30.
64. Stomel RJ, Rasak M, Bates ER. Treatment strategies for acute myocardial infarction complicated by cardiogenic shock in a community hospital. Chest 1994;105:997–1002.
65. Prewitt RM, Gu S, Schick U, et al. Intraaortic balloon counterpulsation enhances coronary thrombolysis induced by intravenous administration of a thrombolytic agent. J Am Coll Cardiol 1994;23:794–8.
66. Moosvi AR, Khaja F, Villanueva L, et al. Early revascularization improves survival in cardiogenic shock complicating acute myocardial infarction. J Am Coll Cardiol 1992;19:907–14.
67. Hibbard MD, Holmes DRJ, Bailey KR, et al. Percutaneous transluminal coronary angioplasty in patients with cardiogenic shock. J Am Coll Cardiol 1992;19:639–46.
68. Holmes DRJ, Califf RM, Van de Werf F, et al. Difference in countries' use of resources and clinical outcome for patients with cardiogenic shock after myocardial infarction: results from the GUSTO trial. Lancet 1997;349: 75–8.
69. DeWood MA, Notske RN, Berg RJ, et al. Medical and surgical management of early Q wave myocardial infarction. I. Effects of surgical reperfusion on survival, recurrent myocardial infarction, sudden death and functional class at 10 or more years of follow-up. J Am Coll Cardiol 1989;14:65–77.
70. Allen BS, Buckberg GD, Fontan FM, et al. Superiority of controlled surgical reperfusion versus percutaneous transluminal coronary angioplasty in acute coronary occlusion. J Thorac Cardiovasc Surg 1993;105:864–79.
71. Mennon V, Sleeper LA, Fincke R, et al. Outcomes with pulmonary artery catheterization in cardiogenic shock [abstract]. J Am Coll Cardiol 1998;31 Suppl A:397A.
72. Tcheng JE, Jackman JD Jr, Nelson CL, et al. Outcome of patients sustaining acute ischemic mitral regurgitation during myocardial infarction. Ann Intern Med 1992;117:18–24.
73. Clements Jr, SD, Story WE, Hurst JW, et al. Ruptured papillary muscle, a complication of myocardial infarction: clinical presentation, diagnosis, and treatment. Clin Cardiol 1985;8:93–103.
74. Kishon Y, Iqbal A, Oh JK, et al. Evolution of echocardiographic modalities in detection of postmyocardial infarction ventricular septal defect and papillary muscle rupture: study of 62 patients. Am Heart J 1993;126:667–75.
75. Gold HK, Leinbach RC, Sanders CA, et al. Intraaortic balloon pumping for ventricular septal defect or mitral regurgitation complicating acute myocardial infarction. Circulation 1973;47:1191–6.
76. Yoran C, Yellin EL, Becker RM, et al. Mechanism of reduction of mitral regurgitation with vasodilator therapy. Am J Cardiol 1979;43:773–7.
77. Pichard AD, Kay R, Smith H, et al. Large V waves in the pulmonary wedge pressure tracing in the absence of mitral regurgitation. Am J Cardiol 1982;50:1044–50.
78. Snyder II, RW, Glamann DB, Lange RA, et al. Predictive value of prominent pulmonary arterial wedge V waves in assessing the presence and severity of mitral regurgitation. Am J Cardiol 1994;73:568–70.
79. Meister SG, Helfant RH. Rapid bedside differentiation of ruptured interventricular septum from acute mitral insufficiency. N Engl J Med 1972;287: 1024–5.
80. Greenberg MA, Gitler B. Left ventricular rupture in a patient with coexisting right ventricular infarction. N Engl J Med 1983;309:539–42.
81. Oliva PB, Hammill SC, Edwards WD. Cardiac rupture, a clinically predictable complication of acute myocardial infarction: report of 70 cases with clinicopathologic correlations. J Am Coll Cardiol 1993;22:720–6.
82. Zehender M, Kaspar W, Kauder E, et al. Right ventricular infarction as an independent predictor of prognosis after acute inferior myocardial infarction. N Engl J Med 1993;328:981–8.
83. Shah PK, Maddahi J, Staniloff HM, et al. Variable spectrum and prognostic implications of left and right ventricular ejection fractions in patients with and without clinical heart failure after acute myocardial infarction. Am J Cardiol 1986;58:387–93.
84. Cresci SG, Goldstein JA. Hemodynamic manifestations of ischemic right heart dysfunction. Cathet Cardiovasc Diagn 1992;27:28–33.
85. Berger PB, Ryan TJ. Inferior myocardial infarction: high-risk subgroups. Circulation 1990;81:401–11.
86. Goldstein JA, Barzilai B, Rosamond TL, et al. Determinants of hemodynamic compromise with severe right ventricular infarction. Circulation 1990;82:359–68.
87. Goldstein JA, Tweddell JS, Barzilai B, et al. Importance of left ventricular function and systolic ventricular interaction to right ventricular performance during acute right heart ischemia. J Am Coll Cardiol 1992;19:704–11.

88. Dell'Italia LJ, Starling MR, Blumhardt R, et al. Comparative effects of volume loading, dobutamine, and nitroprusside in patients with predominant right ventricular infarction. Circulation 1985;72:1327–35.
89. Kinn JW, Ajluni SC, Samyn JG, et al. Rapid hemodynamic improvement after reperfusion during right ventricular infarction. J Am Coll Cardiol 1995;26:1230–4.
90. Bowers TR, O'Neil WW, Grines C, et al. Effect of reperfusion on biventricular function and survival after right ventricular infarction. N Engl J Med 1998;338:933–40.
91. Goldstein JA, Vlahakes GJ, Verrier ED, et al. Volume loading improves low cardiac output in experimental right ventricular infarction. J Am Coll Cardiol 1983;2:270–8.
92. Bailey JM, Levy JH, Kopel MA, et al. Relationship between clinical evaluation of peripheral perfusion and global hemodynamics in adults after cardiac surgery. Crit Care Med 1990;18:1353–6.
93. Sanchez R, Wee M. Perioperative myocardial ischemia: early diagnosis using the pulmonary artery catheter. J Cardiothorac Vasc Anesth 1991;5: 604–7.
94. Lehot JJ, Carre R, Cottet-Emard JM, et al. Does Swan-Ganz catheter help in weaning from controlled ventilation after mitral valve replacement? Presse Med 1988;17:2121–4.
95. Pierson MG, Funk M. Technology versus clinical evaluation for fluid management decisions in CABG patients. Image J Nurs Sch 1989;21:192–5.
96. Moore CH, Lombardo TR, Allums JA, et al. Left main coronary artery stenosis: hemodynamic monitoring to reduce mortality. Ann Thorac Surg 1978;26:445–51.
97. Bashein G, Johnson PW, Davis KB, et al. Elective coronary bypass surgery without pulmonary artery catheter monitoring. Anesthesiology 1985;63: 451–4.
98. Tuman KJ, McCarthy RJ, Spiess BD, et al. Effect of pulmonary artery catheterization on outcome in patients undergoing coronary artery surgery. Anesthesiology 1989;70:199–206.
99. Pearson KS, Gomez MN, Moyers JR, et al. A cost/benefit analysis of randomized invasive monitoring for patients undergoing cardiac surgery. Anesth Analg 1989;69:336–41.
100. D'Alonzo GE, Barst RJ, Ayres SM, et al. Survival in patients with primary pulmonary hypertension: results from a national prospective registry. Ann Intern Med 1991;115:343–9.
101. Rubin LJ. Primary pulmonary hypertension. N Engl J Med 1997;336:111–7.
102. Rich S, Dantzker DR, Ayres SM, et al. Primary pulmonary hypertension: a national prospective study. Ann Intern Med 1987;107:216–23.
103. Sandoval J, Bauerle O, Palomar A, et al. Survival in primary pulmonary hypertension. Validation of a prognostic equation. Circulation 1994;89: 1733–44.
104. Hopkins WE, Ochoa LL, Richardson GW, et al. Comparison of the hemodynamics and survival of adults with severe primary pulmonary hypertension or Eisenmenger syndrome. J Heart Lung Transplant 1996;15: 100–5.
105. Galie N, Ussia G, Passarelli P, et al. Role of pharmacologic tests in the treatment of primary pulmonary hypertension. Am J Cardiol 1995;75:55A–62A.
106. Houde C, Bohn DJ, Freedom RM, et al. Profile of paediatric patients with pulmonary hypertension judged by responsiveness to vasodilators. Br Heart J 1993;70:461–8.
107. Olschewski H, Walmrath D, Schermuly R, et al. Aerosolized prostacyclin and iloprost in severe pulmonary hypertension. Ann Intern Med 1996;124: 820–4.
108. Channick RN, Newhart JW, Johnson FW, et al. Pulsed delivery of inhaled nitric oxide to patients with primary pulmonary hypertension: an ambulatory delivery system and initial clinical tests. Chest 1996;109:1545–9.
109. Rich S, Brundage BH. High-dose calcium channel-blocking therapy for primary pulmonary hypertension: evidence for long-term reduction in pulmonary arterial pressure and regression of right ventricular hypertrophy. Circulation 1987;76:135–41.
110. Weir EK, Rubin LJ, Ayres SM, et al. The acute administration of vasodilators in primary pulmonary hypertension: experience from the National Institutes of Health Registry on Primary Pulmonary Hypertension. Am Rev Respir Dis 1989;140:1623–30.
111. Palevsky HI, Schloo BL, Pietra GG, et al. Primary pulmonary hypertension: vascular structure, morphometry, and responsiveness to vasodilator agents. Circulation 1989;80:1207–21.
112. Palevsky HI, Long W, Crow J, et al. Prostacyclin and acetylcholine as screening agents for acute pulmonary vasodilator responsiveness in primary pulmonary hypertension. Circulation 1990;82:2018–26.
113. Packer M, Medina N, Yushak M. Adverse hemodynamic and clinical effects of calcium channel blockade in pulmonary hypertension secondary to obliterative pulmonary vascular disease. J Am Coll Cardiol 1984;4:890–901.
114. Morgan JM, McCormack DG, Griffiths MJ, et al. Adenosine as a vasodilator in primary pulmonary hypertension. Circulation 1991;84:1145–9.
115. Palevsky HI, Fishman AP. The management of primary pulmonary hypertension. JAMA 1991;265:1014–20.
116. Raffy O, Azarian R, Brenot F, et al. Clinical significance of the pulmonary vasodilator response during short-term infusion of prostacyclin in primary pulmonary hypertension. Circulation 1996;93:484–8.
117. Ogata M, Ohe M, Shirato K, et al. Effects of a combination therapy of anticoagulant and vasodilator on the long-term prognosis of primary pulmonary hypertension. Jpn Circ J 1993;57:63–9.
118. Barst RJ, Rubin LJ, McGoon MD, et al. Survival in primary pulmonary hypertension with long-term continuous intravenous prostacyclin. Ann Intern Med 1994;121:409–15.
119. Barst RJ, Rubin LJ, Long WA, et al. A comparison of continuous intravenous epoprostenol (prostacyclin) with conventional therapy for primary pulmonary hypertension: the Primary Pulmonary Hypertension Study Group. N Engl J Med 1996;334:296–302.
120. Cremona G, Higenbottam T. Role of prostacyclin in the treatment of primary pulmonary hypertension. Am J Cardiol 1995;75:67A–71A.
120a. McLaughlin VV, Genthner DE, Panella MM, Rich S. Reduction in pulmonary vascular resistance with long-term epoprostenol (prostacyclin) therapy in primary pulmonary hypertension. 1998;338:273–7.
121. Matthay MA, Chatterjee K. Bedside catheterization of the pulmonary artery: risks compared with benefits. Ann Intern Med 1988;109:826–34.
122. Damen J, Bolton D. A prospective analysis of 1,400 pulmonary artery catheterizations in patients undergoing cardiac surgery. Acta Anaesthesiol Scand 1986;30:386–92.
123. Lopez-Sendon J, Lopez de Sa E, Gonzalez Maqueda I, et al. Right ventricular infarction as a risk factor for ventricular fibrillation during pulmonary artery catheterization using Swan-Ganz catheters. Am Heart J 1990;119:207–9.
124. Shaw TJ. The Swan-Ganz pulmonary artery catheter. Incidence of complications, with particular reference to ventricular dysrhythmias, and their prevention. Anaesthesia 1979;34:651–6.
125. Iberti TJ, Benjamin E, Gruppi L, et al. Ventricular arrhythmias during pulmonary artery catheterization on the intensive care unit: prospective study. Am J Med 1985;78:451–4.
126. Sprung CL, Elser B, Schein RM, et al. Risk of right bundle-branch block and complete heart block during pulmonary artery catheterization. Crit Care Med 1989;17:1–3.
127. Davidson DJ, Harrison JK. Basic catheter techniques. Invasive Cardiol 1989;109–34.
128. Hardy JF, Morissette M, Taillefer J, et al. Pathophysiology of rupture of the pulmonary artery by pulmonary artery balloon-tipped catheters. Anesth Analg 1983;62:925–30.
129. Pellegrini RV, Marcelli G, Di Marco RF, et al. Swan-Ganz catheter induced pulmonary hemorrhage. J Cardiovasc Surg (Torino) 1987;28: 646–9.
130. Carlson TA, Goldenberg IF, Murray PD, et al. Catheter-induced delayed recurrent pulmonary artery hemorrhage. Intervention with therapeutic embolism of the pulmonary artery. JAMA 1989;261:1943–5.
131. Pape LA, Haffajee CI, Markis JE, et al. Fatal pulmonary hemorrhage after use of the flow-directed balloon-tipped catheter. Ann Intern Med 1979;90: 344–7.
132. Chastre J, Cornud F, Bouchama A, et al. Thrombosis as a complication of pulmonary-artery catheterization via the internal jugular vein: prospective evaluation by phlebography. N Engl J Med 1982;306:278–81.
133. Pace NL, Horton W. Indwelling pulmonary artery catheters: their relationship to aseptic thrombotic endocardial vegetations. JAMA 1975;233:893–4.
134. Boyd KD, Thomas SJ, Gold J, et al. A prospective study of complications of pulmonary artery catheterizations in 500 consecutive patients. Chest 1983;84:245–9.
135. Sise MJ, Hollingsworth P, Brimm JE, et al. Complications of the flow-directed pulmonary artery catheter: a prospective analysis in 219 patients. Crit Care Med 1981;9:315–8.

136. Rowley KM, Clubb KS, Smith GJ, et al. Right-sided infective endocarditis as a consequence of flow-directed pulmonary-artery catheterization: a clinicopathological study of 55 autopsied patients. N Engl J Med 1984;311: 1152–6.
137. Sasaki TM, Panke TW, Dorethy JF, et al. The relationship of central venous and pulmonary artery catheter position to acute right-sided endocarditis in severe thermal injury. J Trauma 1979;19:740–3.
138. Singh S, Nelson N, Acosta I, et al. Catheter colonization and bacteremia with pulmonary and arterial catheters. Crit Care Med 1982;10:736–9.
139. McDaniel DD, Stone JG, Faltas AN, et al. Catheter-induced pulmonary artery hemorrhage. Diagnosis and management in cardiac operations. J Thorac Cardiovasc Surg 1981;82:1–4.
140. Wiedemann HP, Matthay MA, Matthay RA. Cardiovascular-pulmonary monitoring in the intensive care unit (part 2). Chest 1984;85:656–68.
141. Mermel LA, Maki DG. Infectious complications of Swan-Ganz pulmonary artery catheters: pathogenesis, epidemiology, prevention, and management. Am J Respir Crit Care Med 1994;149:1020–36.
142. Pearson AC, Castello R, Labovitz AJ. Safety and utility of transesophageal echocardiography in the critically ill patient. Am Heart J 1990;119:1083–9.
143. Heidenreich PA, Stainback RF, Redberg RF, et al. Transesophageal echocardiography predicts mortality in critically ill patients with unexplained hypotension. J Am Coll Cardiol 1995;26:152–8.
144. Khoury AF, Afridi I, Quinones MA, et al. Transesophageal echocardiography in critically ill patients: feasibility, safety, and impact on management. Am Heart J 1994;127:1363–71.
145. Kaul S, Stratienko AA, Pollock SG, et al. Value of two-dimensional echocardiography for determining the basis of hemodynamic compromise in critically ill patients: a prospective study. J Am Soc Echocardiogr 1994;7:598–606.
146. Peels CH, Visser CA, Kupper AJ, et al. Usefulness of two-dimensional echocardiography for immediate detection of myocardial ischemia in the emergency room. Am J Cardiol 1990;65:687–91.
147. Singh S, Wann LS, Schuchard GH, et al. Right ventricular and right atrial collapse in patients with cardiac tamponade—a combined echocardiographic and hemodynamic study. Circulation 1984;70:966–71.
148. Appleton CP, Hatle LK, Popp RL. Cardiac tamponade and pericardial effusion: respiratory variation in transvalvular flow velocities studied by Doppler echocardiography. J Am Coll Cardiol 1988;11:1020–30.
149. Smyllie JH, Sutherland GR, Geuskens R, et al. Doppler color flow mapping in the diagnosis of ventricular septal rupture and acute mitral regurgitation after myocardial infarction. J Am Coll Cardiol 1990;15:1449–55.
150. Stewart WJ, Currie PJ, Salcedo EE, et al. Evaluation of mitral leaflet motion by echocardiography and jet direction by Doppler color flow mapping to determine the mechanisms of mitral regurgitation. J Am Coll Cardiol 1992;20:1353–61.
151. Buda AJ. The role of echocardiography in the evaluation of mechanical complications of acute myocardial infarction. Circulation 1991;84:I109–21.
152. Helmcke F, Mahan III, EF, Nanda NC, et al. Two-dimensional echocardiography and Doppler color flow mapping in the diagnosis and prognosis of ventricular septal rupture. Circulation 1990;81:1775–83.
153. Valdes-Cruz LM, Horowitz S, Mesel E, et al. A pulsed Doppler echocardiographic method for calculation of pulmonary and systemic flow: accuracy in a canine model with ventricular septal defect. Circulation 1983;68: 597–602.
154. Lopez-Sendon J, Gonzalez A, Lopez de Sa E, et al. Diagnosis of subacute ventricular wall rupture after acute myocardial infarction: sensitivity and specificity of clinical, hemodynamic and echocardiographic criteria. J Am Coll Cardiol 1992;19:1145–53.
155. Gatewood Jr, RP, Nanda NC. Differentiation of left ventricular pseudoaneurysm from true aneurysm with two dimensional echocardiography. Am J Cardiol 1980;46:869–78.
156. Lascault G, Reeves F, Drobinski G. Evidence of the inaccuracy of standard echocardiographic and angiographic criteria used for the recognition of true and "false" left ventricular inferior aneurysms. Br Heart J 1988;60: 125–7.
157. Sutherland GR, Smyllie JH, Roelandt JR. Advantages of colour flow imaging in the diagnosis of left ventricular pseudoaneurysm. Br Heart J 1989;61:59–64.
158. D'Arcy B, Nanda NC. Two-dimensional echocardiographic features of right ventricular infarction. Circulation 1982;65:167–73.
159. Rietveld AP, Merrman L, Essed CE, et al. Right to left shunt with severe hypoxemia at the atrial level in a patient with hemodynamically important right ventricular infarction. J Am Coll Cardiol 1983;2:776–9.
160. Dabaghi SF, Rokey R, Rivera JM, et al. Comparison of echocardiographic assessment of cardiac hemodynamics in the intensive care unit with right-sided cardiac catheterization. Am J Cardiol 1995;76:392–5.
161. Brunazzi MC, Chirillo F, Pasqualini M, et al. Estimation of left ventricular diastolic pressures from precordial pulsed-Doppler analysis of pulmonary venous and mitral flow. Am Heart J 1994;128:293–300.
162. Vanoverschelde JJ, Robert AR, Gerbaux A, et al. Noninvasive estimation of pulmonary arterial wedge pressure with Doppler transmitral flow velocity pattern in patients with known heart disease. Am J Cardiol 1995;75:383–9.
163. Yock PG, Popp RL. Noninvasive estimation of right ventricular systolic pressure by Doppler ultrasound in patients with tricuspid regurgitation. Circulation 1984;70:657–62.
164. Currie PJ, Seward JB, Chan KL, et al. Continuous wave Doppler determination of right ventricular pressure: a simultaneous Doppler-catheterization study in 127 patients. J Am Coll Cardiol 1985;6:750–6.
165. Vanoverschelde JJ, Raphael DA, Robert AR, et al. Left ventricular filling in dilated cardiomyopathy: relation to functional class and hemodynamics. J Am Coll Cardiol 1990;15:1288–95.
166. Giannuzzi P, Imparato A, Temporelli PL, et al. Doppler-derived mitral deceleration time of early filling as a strong predictor of pulmonary capillary wedge pressure in postinfarction patients with left ventricular systolic dysfunction. J Am Coll Cardiol 1994;23:1630–7.
167. Rossvoll O, Hatle LK. Pulmonary venous flow velocities recorded by transthoracic Doppler ultrasound: relation to left ventricular diastolic pressures. J Am Coll Cardiol 1993;21:1687–96.
168. Labovitz AJ, Buckingham TA, Habermehl K, et al. The effects of sampling site on the two-dimensional echo-Doppler determination of cardiac output. Am Heart J 1985;109:327–32.
169. Moulinier L, Venet T, Schiller NB, et al. Measurements of aortic blood flow by Doppler echocardiography: day to day variability in normal subjects and applicability in clinical research. J Am Coll Cardiol 1991;17:1326–33.
170. Kitabatake A, Ito H, Inoue M, et al. A new approach to noninvasive evaluation of aortic regurgitant fraction by two-dimensional Doppler echocardiography. Circulation 1985;72:523–9.
171. Sanders SP, Yeager S, Williams RG. Measurement of systemic and pulmonary blood flow and QP/QS ratio using Doppler and two-dimensional echocardiography. Am J Cardiol 1983;51:952–6.
172. Thys DM, Abel M, Bollen BA, et al. Practice guidelines for perioperative transesophageal echocardiography: a report by the American Society of Anesthesiologists and the Society of Cardiovascular Anesthesiologists Task Force on Transesophageal Echocardiography. Anesthesiology 1996;84: 986–1006.
173. Reichert CL, Visser CA, Koolen JJ, et al. Transesophageal echocardiography in hypotensive patients after cardiac operations: comparison with hemodynamic parameters. J Thorac Cardiovasc Surg 1992;104:321–6.
174. Chan KL. Transesophageal echocardiography for assessing cause of hypotension after cardiac surgery. Am J Cardiol 1988;62:1142–3.
175. Gold MR, Siegel JE, Russell LB, et al. Cost-effectiveness in Health and Medicine. New York: Oxford University Press, 1996.
176. Spackman TN. A theoretical evaluation of cost-effectiveness of pulmonary artery catheters in patients undergoing coronary artery surgery. J Cardiothorac Vasc Anesth 1994;8:570–6.
177. Spodick DH. Flow-directed pulmonary artery catheterization: moratorium vs clinical trial. Chest 1989;95:489–90.